ON THE 'VOMITING SICKNESS' OF JAMAICA

BY

H. HAROLD SCOTT, M.D. Lond., D.P.H. Hons.

GOVERNMENT BACTERIOLOGIST; PATHOLOGIST TO THE PUBLIC GENERAL HOSPITAL, KINGSTON, JAMAICA

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I. INTRODUCTION

The so-called Vomiting Sickness of Jamaica has been a veritable scourge in this island for many years past. It rages during the cooler months of the year, starting towards the end of November or beginning of December and continuing its ravages to the end of March or well into April. The mortality rate is very high, and in some years it takes a toll of deaths which runs into hundreds. During the last season, December, 1914 to April, 1915, the prevalence has been exceptionally widespread and severe, and I had the good fortune to be able to study on the spot a very fatal outbreak on the north side of the island. As a result of that investigation I have undertaken certain experimental work—described in this paper—to clear up the mysterious question of the causation of this peculiar and interesting disease.

II. HISTORICAL

The earliest recorded statements concerning this peculiar affection which I have been able to find are those made in January, 1900, by Dr. R. G. S. Bell, District Medical Officer of May Pen, and by Dr. H. G. Tillman, at Vere, in February of the same year.

Other records, between 1900 and 1905, were given by Drs. Ker, J. A. L. Calder, Edwards, Turton, Cooke, A. W. Thomson, and Earle.

During the following year (1905-6) the disease was much less extensive and prevailed for a shorter period, viz., December to February. It occurred most at Newport, a district which had not been mentioned in the previous outbreak.

The year 1906 is an important one in the history of the affection, for a letter was addressed to the authorities of other West Indian Islands, describing the disease and enquiring whether anything similar was prevalent in those localities. Replies were mainly in the negative; two only are quoted as of interest.

(i) Dr. Carlos Finlay (1906) mentions an outbreak which he calls cerebro-spinal meningitis, with five fatal cases, occurring in the troops of an American regiment near Mariano in Cuba. This is certainly not the same as the vomiting sickness of Jamaica, for it attacked adults only and did not recur after the single outbreak in 1899. Also, as will be seen later, the symptoms and course of cerebro-spinal meningitis differ from those of typical vomiting sickness.

(ii) The Consul-General in Haiti (1906) wrote: 'There is a 'disease somewhat similar in this island. It has not, however, 'proved to be particularly dangerous. It generally happens 'during the months of February, March, and April, when there 'is a difference in temperature by day and by night. It can 'best be described as severe bowel troubles, together with high 'fever and vomiting. Europeans suffer principally from it, and 'natives to a much less extent. Children are apt to suffer from 'it, but there does not appear to be any high mortality from it. 'The attack (acute stage) lasts on an average from three to five 'days, when improvement sets in with rapid convalescence.'

It will be seen from a perusal of the following pages that this

condition is quite different from the vomiting sickness of Jamaica. Briefly, the differences may be tabulated thus:—

Haitian Disease	*Vomiting Sickness*
1. Bowel troubles.	1. No bowel troubles, as a rule.
2. High fever.	2. Hardly any fever; temperature may rise to 99·4°, seldom reaches 100° F.
3. Europeans principally attacked, natives less.	3. Europeans practically never, natives about 99 per cent.
4. Mortality not high.	4. Mortality about 80 per cent.
5. Attack (acute stage) lasts three to five days.	5. Death usually in 15 hours, may occur within an hour. If attack lasts 24 hours, there is nearly always recovery.

This is mentioned in some detail because one of the chief points to be explained (see later) is the confinement of the disease, so far as is known, to Jamaica.

Replies were also received from Cuba, Nassau, Trinidad, Grenada, Barbadoes, Santi Domingo, British Guiana, Antigua, St. Vincent, St. Lucia and Porto Rico; so that we may infer that the inhabitants of the other islands do not suffer from any disease corresponding to the vomiting sickness.

In the 1907 report it is noted that there had been that year 'more than ordinarily severe outbreaks of this disease.'

The reports for 1908 and 1909 give very meagre statements as regards this disease.

In the 1909 report there are only casual remarks upon the disease.

In 1910 the disease was fairly widespread and severe.

The following year, 1910-1911, must be dealt with in greater detail, since, in consequence of communications from His Excellency, the Governor, Sir Sydney Olivier, the Research Committee of the Colonial Office selected Captain (now Major) T. J. Potter, R.A.M.C., to carry out investigations into this disease. He arrived on Christmas Day, 1910, and remained in the island till August, 1911. In consequence of his being present and knowing that a report would follow, the various medical officers made but very meagre remarks on the subject for that year.

Potter's report was published in 1912, and he was of the opinion that the majority of deaths ascribed to the so-called vomiting sickness were due to yellow fever.

Eighteen months ago (February, 1914), when writing on this same question, I stated (Scott, 1914): 'Of nearly 200 cases reported 'to me in detail, only two had any vomiting so described, and 'amongst those seen by me personally I have never met with a case 'in which the vomit was black. . . . The vomitus in all that 'I have seen has been in the main mucoid, watery, or frothy, while 'very occasionally, if there has been much straining or retching, it 'may be pinkish from admixed blood.'

Dr. Seidelin (1913) was struck by the rarity of black vomit.

We now come to what may be called the 'meningitis era' of the history of the investigation, in reality a recrudescence of the older theory that vomiting sickness and cerebro-spinal meningitis were one and the same. This period was intermediate between the investigation of Captain Potter and that of Dr. Seidelin.

Though cases of meningitis occur amongst those reported as vomiting sickness, the two diseases are not, I believe, identical. After the former have been excluded, a large proportion still remains unaccounted for, so far as actual knowledge of the cause goes at present.

Meanwhile, in January, 1913, Dr. Harald Seidelin arrived from England, having been sent out by the Liverpool School of Tropical Medicine to investigate the disease. His report (Seidelin, 1913) with notes and analyses of 62 cases appeared in the *Annals of Tropical Medicine and Parasitology*, Vol. VII, pp. 377-478.

The great value of this report consists in the fact of its containing such excellent accounts of the morbid anatomy of the disease. The post-mortem appearances, both macroscopical and microscopical, are carefully described in detail.

In the year subsequent to that of Seidelin's investigations very few cases occurred.

III. PERSONAL OBSERVATIONS ON THE MONTEGO BAY OUTBREAK, FEBRUARY, 1915

Such was the state of the question at the beginning of 1915. It will be seen that various diagnoses had been given from time to time—gastritis, gastro-enteritis, worms, malaria, pneumonia, cerebro-spinal meningitis, yellow fever, and so on.

In the sense that possibly all these had at one time or another

been included under the general heading of vomiting sickness we might say, in the words of Kipling, 'Every single one of them is right.' As has been already stated, during the cooler months, any case of a child who dies after having vomited, unless there is some very definite cause, is diagnosed as vomiting sickness; while others, dying from some obscure condition, if vomiting has not occurred, are diagnosed as 'vomiting sickness without vomiting.'

In short, the name of 'vomiting sickness' has been used as successfully as 'charity' in covering a multitude of sins. Apart from those mentioned above, among the number reported to me as cases of vomiting sickness I may mention such as proved to be marasmus, intussusception, status epilepticus, cerebral haemorrhage, pregnancy. Truly a chaotic state of things this, when the epidemic of 1915 began.

During this period the disease was very rife, more widespread and more prolonged. An exceptional opportunity of studying the condition on the spot arose in February, when Dr. Thomson, the District Medical Officer of Montego Bay, reported a severe outbreak with eighteen deaths in two days. I went immediately and stayed in the district, and was able to see several cases, some of them almost from start to finish. In company with the District Medical Officer, who had kept records of most of the cases, I was enabled to interview survivors or the relatives of those who had died. I visited the huts where the cases had occurred, and performed autopsies on all who died during my stay in the neighbourhood. I obtained detailed notes of thirty-two cases of true vomiting sickness, nearly every one conforming to the standard type of the disease. Three others were reported as such, but one turned out to be acute haemorrhagic pancreatitis, one was a case of ordinary infantile convulsions without the usual accompanying symptoms of vomiting sickness, the third was a patient suffering from ordinary malaria of fairly long standing, not an acute case at all. *Plasmodium falciparum* was seen in her blood, and she made a complete recovery on the usual antimalarial lines of treatment.

Since this outbreak and the investigations resulting from it appear to furnish the key to the problem, and as nearly every case brings a certain weight of corroborative evidence, the cases must be given in some detail; detail, that is, as regards history only, as I do not

wish to burden this section with pathological findings, which have their place subsequently.

Case 1. S.M., female, 12 years of age; living at 'Retirement.' On February 16th went to school perfectly well. About 4 p.m. complained of pain in the stomach, and vomited three times. Felt better and tried to get home, but on the way felt ill and again vomited, and finally reached home between 7.30 and 8 p.m. Shortly afterwards she passed into a state of coma and died about midnight. Nature of previous meal not known; many ackee trees with ripe fruit in the yard.

The following eleven cases occurred at 'Salt Spring':—

Case 2. D.C., female, 6 years. Well on going to bed on February 16th. At 5 a.m., 17th, vomited twice, but did not complain of any pain. Felt sick all day 17th; at 6 a.m., 18th, after a fairly good night, vomiting started again; almost immediately afterwards she had a convulsion and passed into a state of coma, in which she died at 2 p.m.

Her food consisted of yam, banana, and 'probably ackee,' according to the mother. There were many in the yard, but the mother would not say for certain whether the child had eaten them.

Case 3. N.R.D., male, 10 years. At 5 p.m., February 17th, complained of pain in the stomach, and began to vomit; continued to do so until 1 a.m., 18th, when convulsions set in, followed by coma which lasted till death, at noon.

The food taken at 3 p.m., 17th, had consisted of yam, salt-fish, ackee, and bananas, all boiled together. The ackee was mostly removed and eaten by the older members of the family (Nos. 5, 6, and 7), leaving the 'soup' or 'pot-water' for this child, and the one whose case follows.

Case 4. M.C., female, 13 years, cousin to the last, and living in the same hut. Vomiting began about the same time, 5 p.m., and ceased at midnight; returned about noon, 18th, and lasted till 3 p.m. There were no convulsions, but coma with restlessness; death took place at 3 a.m., 19th.

Food: part of the same meal as that of the last patient, viz., the 'soup' of the yam, bananas, ackee, etc., which had been boiled together.

Case 5. R.C., female, 26 years, mother of N.R.D. (No. 3), and aunt of M.C. (No. 4). Vomiting began at 5 p.m., 17th, and continued all night. It ceased in the early hours of the 18th. Feeling better she took some more of the same articles during the day as had been boiled for the family on the 17th. The vomiting started again the same evening and continued during the following day. This patient recovered.

Case 6. J.J., female, 65 years; lived with the last three, and was the mother of R.C., and grandmother of N.R.D. Vomiting began about 8 p.m., 18th, and continued till 2 a.m., 19th. During the 19th she vomited at intervals, but eventually recovered.

This woman partook of the same meal as the others (yam, bananas, salt-fish, and ackee).

Case 7. B.J., female, 21 years; another daughter of the last. Vomiting began during the evening of the 18th and continued at intervals for 24 hours. She also recovered.

Her food was the same as that of the four previous cases.

An important point to note in this series of cases is that the same

food was used by all; the older members ate the solid and recovered, the younger were given the 'soup' or 'pot-water' and died. Also R. C. (No. 5) showed a return of the symptoms on again partaking of a similar diet.

Case 8. F.W., female, 8 years. On February 17th she had a meal of 'yam, banana, pumpkin, and possibly ackee' (parents' statement) all boiled together. There were many ackees in the yard. Vomiting began the same evening, and shortly afterwards convulsions and coma supervened, and she remained comatose till death, at noon on the 18th.

Case 9. H.W., male, 1 year and 9 months; brother to the last-named. Was apparently quite well on going to bed on the 17th. About dawn, 18th, complained of pain in the stomach: soon after was attacked by convulsions, became comatose, and died at 8 a.m. His food was the 'soup' or 'pot-water,' i.e., the gravy or liquid from boiling the above articles of food.

Case 10. P.C., female, 25 years; mother of the last two. Meal on the 17th consisted of yam, bananas, pumpkin, and ackee. Early on the 18th, 'about dawn,' suffered from griping pains, vomiting and diarrhoea. These symptoms continued all day, and she felt so ill that she could not even attend to the two children who were dying. The symptoms subsided the next day, and she rapidly recovered.

The history as regards the nature of the food in this case was quite definite, and tends to prove the presence of ackee in the meal of the two children. It is incredible that a poor woman, such as this was, would cook two similar breakfasts, one for the children and another for herself, one of yams, bananas, and pumpkin, and the other of yams, bananas, pumpkin, and ackees. Probably, nay, almost certainly, all four ingredients were cooked together, as is customary, the ackees being picked out and eaten by the mother, the more solid residue by the older child, the 'pot-water' being given to the younger.

Note the graded acuteness of the illness: the mother, who had the solid ackees, was seriously ill for thirty-six hours or so, and recovered; the older child partook of the other ingredients, with, of course, some of the absorbed 'pot-water,' was ill for about sixteen hours, and then died; the younger, who had the 'pot-water' only, was acutely ill, and died in about two hours.

Case 11. R.M., female, 4 years. At 10 a.m., February 18th, was seized with convulsions (there may have been vomiting previously, but this was uncertain), rapidly became comatose, and died at 2 p.m.

This case was reported as one of vomiting sickness and is, therefore, included here. The post-mortem findings were those of vomiting sickness, but otherwise it might be merely a case of infantile convulsions of gastric or intestinal origin associated with worms, for many Ascarides were present.

Food: uncertain; said to be 'pap,' but the child was running about, and there were many ackees in the yard, and she could easily have picked up an unsound one and have eaten it.

Case 12. I.F., female, 4 years. Apparently well at 3 p.m., February 20th, when she lay down and went to sleep. An hour later her mother tried to awaken her, but could not. On stimulation the child seemed to revive a little, but

shortly afterwards there was twitching and a slight convulsion, with coma, which deepened till death, at 1 p.m. on February 21st.

The food consisted of yam, bananas, and 'possibly ackee.' The mother was 'not quite certain' whether the patient ate any. There were large numbers in the yard.

The following case occurred at Green Pond, Portobello, near Salt Spring:—

Case 13. A.R., female, 2 years 10 months. At 11 a.m., February 24th, a meal, consisting of pumpkin, yam, peas, and ackees, all boiled together, was prepared for the family. This child was given the 'pot-water.' Two hours later she complained of pain in the stomach, and at 1 p.m., after vomiting twice, she went to lie down. Vomiting increased in frequency till 2 p.m., when muscular twitchings and convulsive attacks supervened, the child lost consciousness, and died comatose at 5 p.m.

The next six cases occurred at Montego Bay itself.

Case 14. G.G., male, 8 years. At noon on February 17th a meal was prepared, which consisted of yam, bananas, and ackees, all boiled together. The parents stated that they themselves ate the ackees, while the patient and his sister (No. 15) were given the 'pot-water,' and some of the other ingredients. He started to vomit at 3 p.m., and continued to do so till 9 p.m., when convulsions came on, succeeded by coma, which terminated fatally at 2 a.m., 18th.

Case 15. D.G., female, 11 years; sister to the last. Vomiting started during the night of the 17th and continued till the morning when convulsions and coma came on, and death occurred at 10 a.m., 18th.

The history as regards food is the same as the last.

These two patients had accompanied their parents to their provision ground, a mile away, where the meal was cooked.

Case 16. W.G., male, 9 years; and

Case 17. A.G., female, 4 years; brother and sister respectively of the last two. These were away from the rest of the family during the day. The history of both was the same. Vomiting, not very severe, and not accompanied by convulsions or loss of consciousness, began in the morning of the 18th. Both recovered in 24 hours.

The parents were not very sure about the food in these two cases, while they were away. There were many ackee trees in the yard, where they stayed all day, and also the parents thought that they had had some of the remains of the food left over from the meal described above—yam, bananas, and ackees.

These four cases are interesting. They all belong to the same family. Two undoubtedly were given ackee or ackee-water (pot-water); the other two had probably eaten some. Those who were known to have had the 'soup' or 'pot-water' died, while the others suffered from vomiting, but recovered.

Case 18. B.R., female, 14 years 11 months. Started to vomit at 4 p.m. 17th, and continued to do so during the night. In the course of the following morning she became unconscious, and remained comatose till death on the 19th.

As regards the question of the food in this case. All the statement obtainable

from the mother was 'ordinary food,' and when pressed for details would only say 'yam and beef.' This was exceedingly unlikely; the place was poor, the child emaciated, beef would be a great luxury, and in such a state of poverty ackees would be certainly used as they were plentiful in the yard. Moreover, 'methought the lady did protest too much' against the idea of the girl having eaten any.

Case 19. P.R., male, 6 years. At 5 p.m., February 24th, was given a dinner of salt fish and ackee. At 7 p.m. he suddenly began to vomit, but did not complain of any pain. He vomited up ackee. During the night he improved, but at 5 a.m. he had a fit and became unconscious. He continued in this state, but with deepening coma, till death at 3 p.m.

The next twelve cases occurred at 'Granville,' another sub-district of Montego Bay.

Case 20. W.B.H., male, 5 years. Early on February 18th he began to vomit, but made no complaint of pain. The vomiting continued for an hour, when convulsions supervened, and the child passed into a state of coma, and died about 4 hours after the onset.

Food: see after next case.

Case 21. G.S.H., male, 2 years 10 months; brother to the last. Similar history, but the duration of the illness was 5 hours.

Both children spent the previous day with their grandmother. This woman stated that she gave them yam and bananas, but there were many ackee trees with ripe fruit in the yard, and many ackees were lying on the ground. She denied having given any to the children, but owned that they might easily have picked them up and eaten them.

I am inclined to think that this is more likely, because the delay in onset would then be explained by the fact that only a small part of the fruit might be toxic (some soft part, for example), and being solid took longer to be absorbed. There was, it will be noted, very little local action; there was no complaint of abdominal pain, but almost entirely nervous symptoms.

Case 22. A.M.H., female, 8 years; sister to the last two. Began to vomit at 10 a.m., 18th, and continued to vomit for two to three hours. About 1 p.m. convulsions set in, followed almost at once by coma, which lasted till death, between 6 and 7 p.m.

This child had also been for a short time to the grandmother's hut. Her mother stated that she had given her a meal of yam, bananas, salt-fish, and 'possibly a little ackee.' (See next case.)

Case 23. A.H., female, 42 years; mother of the last three. Had a severe attack of vomiting during February 18th, starting at 6 a.m.; she then felt faint and giddy, but had no convulsions, and did not lose consciousness. She made a good recovery.

This woman definitely stated that her food on the day in question, 17th, consisted of yam, bananas, and ackees. Seeing that the daughter, A.M.H., was with her practically the whole day, and took her meal with her, the former almost certainly had ackees too; for, as mentioned in the series 8, 9, and 10, it is most unlikely, to say the least, that the mother would cook two dinners of the same ingredients—yam and bananas—and place ackees in one and not in the other. More probably, as appears to be customary, the mother ate the ackees herself, and the child had some of the yam and bananas with the 'soup' or 'pot-water.'

The latter's attack terminated fatally, while the former suffered from gastric symptoms mainly, with some vertigo, and recovered.

Case 24. J.McB.B., male, 6 years 9 months. On February 17th, was given a meal of yam, bananas, and ackees, boiled up together. Early on the 18th he appeared 'droopy and dull.' During the day the 'soup' of a similar meal was given to him, but he only took a little of it, and the rest of the liquid was thrown away. At 5.30 p.m. vomiting came on, convulsions followed almost at once, with a condition of deepening coma which lasted till death, at 4 p.m., on the 19th.

Case 25. L.R., female, 26 years; mother of the last. Began to vomit at 9 a.m., 19th, and vomited several times between then and 3 p.m. Vomiting ceased then, and she made a good recovery. She had no convulsions and never lost consciousness.

Her food was the same as that just mentioned—yam, bananas, and ackees—of which she took the solid, the liquid being thrown away after being refused by the child. It will be seen that the mother ate the solid ingredients and so much of the liquid as had soaked into them; she vomited and recovered; whereas the child had some of the 'soup' and died.

Case 26. C.S., female, 25 years. Vomited on and off during February 18th, and late in the afternoon was seized with convulsions, and lost consciousness. She remained comatose till death on the following day.

Nothing could be discovered regarding this patient's food, but it may be noted that she lived in the same yard as the case, No. 24, already related, where ackees were numerous.

Case 27. E.B., female, 13 months. Began to vomit in the evening of February 19th; was almost immediately attacked by convulsions, passed into coma, and died at 4 a.m., 20th.

Food: 'not known, probably pap,' (mother's statement). But the child was accustomed to crawl about unattended and unwatched, and there were ackees lying about in the yard.

Case 28. C.W., female, 8 years. Began to vomit at 2 p.m., 20th; vomited matter appeared to consist of yam and banana. About 6 hours later coma supervened, without any convulsion, and she died without regaining consciousness.

Case 29. S.W., female, 6 years; sister to the last. Vomiting started about 3 p.m. the same day, but was not severe. There was no convulsions nor loss of consciousness, and the child recovered within 24 hours.

Nothing could be ascertained with certainty about the nature of the food in either of these cases, as they were not seen till after they had been brought into hospital. I visited the house where they lived, and noticed several ackee trees bearing fruit plentifully in the yard in which the hut was situated.

Case 30. C.Y., female, 10 years. Complained of abdominal pain on the evening of February 22nd, and vomited on and off during the day and night of the 23rd. Early on the 24th the child seemed worse, and at 8 a.m. was seized with a convulsion, became comatose and remained in this condition till death, at 10.30 a.m.

The food on the day preceding the illness consisted of yam, salt-beef, bananas, and ackees. The mother knew for certain that the child had ackees on that day, and 'may have had them later, but was not quite sure.'

Case 31. E.M., female, 25 years. Vomiting on and off, shortly after meals, for a week preceding the date on which I interviewed her, 24th. Her food had consisted of bananas and ackees. She attributed these attacks to 'weakness,'

and on the 23rd she determined to take a larger meal of fish, ackees, and bananas. The vomiting had been much worse since, but she had had no twitchings or fits. When preparing her food, she had always boiled the ingredients up together, but had been careful to throw away the 'pot-water.'

This woman, therefore, has suffered from repeated attacks of vomiting after taking ackees, and a more severe attack had followed a larger meal containing them. She always threw away the water, but some had doubtless been absorbed by the solid food.

The last case occurred at a place called 'Tuckers,' near the district of Granville.

Case 32. P.M., female, between 8 and 9 years of age. About noon on February 23rd, she was given a meal of fish (herring), bananas, and ackees, all boiled together; but, as she said she did not want it, the ackees were removed and the patient drank some of the 'pot-water' or 'soup.' At 2 p.m. she suddenly vomited, after complaining of abdominal pain. The vomiting ceased by 5 p.m., and the patient passed a fair night. At 6.30 a.m., 24th, she again complained of pain, and vomited, so she was brought to the hospital. No further vomiting occurred, the child felt better the same evening, and appeared quite well again by the following day.

This case is a good example of the toxicity of the 'pot-water.'

A brief consideration of the above series of cases may now be given.

For this purpose they may be grouped in the following manner:

1. In sixteen cases, namely, Nos. 3, 4, 5, 6, 7, 10, 13, 14, 15, 19, 23, 24, 25, 30, 31, and 32, there was a definite history of eating ackee or an extract ('soup' or 'pot-water' made with ackees) at the meal preceding the onset of the illness. If No. 22 be included, and there is almost sufficient evidence to warrant such inclusion in this group, there are seventeen out of the thirty-two in which there is no doubt that the attack followed closely on the ingestion of ackees, or an extract from them.

2. Those cases in which there is sufficient evidence to warrant a strong probability that ackees comprised one of the constituents of the meal prior to the onset of the illness. There are six which would come under this heading, namely, Nos. 2, 8, 9, 12, 20, and 21; or, if No. 22 be taken from Group 1, and placed here, there would be seven.

3. This contains only two cases, Nos. 16 and 17, in which there is some evidence pointing to the fact that ackees were eaten, but not sufficiently strong to warrant their inclusion under the foregoing groups.

4. Lastly, there are seven in which no history was elicited of

the eating of ackees, but it must be noticed that in every instance trees bearing ripe fruit were growing in the yards in which the huts were situated, and amongst the poor people it would be most unlikely that the use of a food which was ready to hand, a food of which they all appear to be fond, and which was then ripe, would be avoided, and that at a time when other articles of food are scarce, or at least relatively expensive. The seven included under this category are Nos. 1, 11, 18, 26, 27, 28, and 29.

Briefly, then, we may say that in none of the thirty-two cases could the eating of ackees shortly before the onset of symptoms be definitely excluded; in sixteen the fact was absolutely certain, in seven more it was almost certain, in two more—giving a total of twenty-five—it was probable, while in the remaining seven it was possible.

The eliciting of a history of ackee-eating is not always easy, for, if the native once gets the idea that there is any suspicion of food or other poisoning, he either becomes stolid and unable (?) to grasp the meaning of the simplest question, or states something quite false with the aim of putting the questioner off the scent. Thus, in the case of No. 18, the patient was badly nourished and lived in a poor hut, but surrounded by ackee trees bearing ripe fruit. The mother stated that the child had been given 'just ordinary food,' and when pressed for details would only reply 'yam and beef,' and tried to make me believe that the family lived practically on these two articles always, and protested most vehemently that they would not think of eating any of the ackees which grew so plentifully at their very door.

Again, 'saltfish' is frequently named as an article of diet in the country districts, and in towns such as Kingston 'saltfish and ackee' is a favourite dish with many. In the country parts, however, 'saltfish' may not be fish at all. I had suspicions of this, because when asked what form of salt fish they had had (where this had been named as part of the meal preceding the illness) there was frequently no answer forthcoming. Dr. Thomson, the District Medical Officer, wrote to me on February 26th a letter, from which I quote:—

'I have since learnt, and I have made enquiries myself, from several of the same class of people as those of Salt Spring and Granville, etc., and found that

the statement is true, viz., that these people are in the habit of adding salt to the ackees and boiling it, and then add it to their food, calling it "salt fish." In many instances when they say they had yam, banana, and *salt fish* for breakfast, the salt-fish they refer to is the *salted ackee*.

26.2.15. (Signed) GEO. WM. THOMSON, D.M.O.'

I have since obtained independent confirmation of this fact from quite another part of Jamaica, that many of the peasants are in the habit of boiling up their ackees and adding salt, and designating the result 'saltfish.'

People generally in this island are convinced that ackees, under certain conditions are poisonous; among these conditions may be mentioned:

i. Unopened ackees, and by consequence
ii. Ackees which have not opened naturally, but have been forced after falling unopened.
iii. Fruit gathered from a decayed branch.
iv. Ackees with some soft spot in an otherwise apparently sound fruit.

Among the better classes the ackees are gathered carefully one by one, and none are cooked but such as appear ripe and sound in every respect. Among the poorer people, on the other hand, as exemplified in the districts of Salt Spring, Granville, etc., a boy is sent up the tree to shake the branches; ripe fruit and some unopened and unripe fall together; the former is collected, while the latter is left on the ground. These last in time open, and may then be gathered with the new ripe ackees which fall at the next shaking. Apparently an ackee fruit which has opened in the natural way on the tree, and then is allowed to become over-ripe, or even decayed, is not poisonous (so I am informed: I have not had an opportunity of proving the statement), but an unripe, unopened fruit which becomes 'forced' open, the adult native is suspicious of. Children, naturally, would not make this distinction, hence the great incidence among those of tender age.

Some apparently ripe and wholesome ackees have a soft spot in the otherwise firm, fleshy part of the fruit; whether this is due to primary microbial development, or whether it arises from bruising when shaken from the tree, I cannot say, but the fact remains that if this is noticed the ackee in question is not used for food.

Next, from a careful consideration of the histories of the above

cases, the poison is apparently extracted by boiling the affected fruit with water. Nos. 3, 4, 8, 9, 10, 13, 14, 15 and 24 support this. The bananas, pumpkin, yam and ackee are boiled up together; the parents eat the ackees, while the children partake of the rest of the food, the older ones having more of the solid parts, which naturally have absorbed some of the water, and the younger children are given the 'soup' or 'pot-water.' The degrees of toxicity are varied in such cases, as in the series of the Johnson-Clark family (Nos. 3, 4, 5, 6, and 7), where the adults who ate the ackees were slightly affected, having attacks of vomiting, but recovering; the older child taking the solid part of the residue, with, of course, absorbed watery extract, and dying after thirty-four hours; the younger child, who mainly had the 'soup' or 'pot-water,' died in nineteen hours. The Glenn cases (Nos. 14 and 15), and the Cook-Waite cases (Nos. 8, 9, and 10), are other instances in point.

No. 13, a case of a young girl under three years of age, who only had 'pot-water,' is another example of the toxicity of the extract, death taking place in four to five hours.

Undoubtedly a certain degree of suspicion attaches to the use of the 'pot-water' as an article of diet, for, if the family is small and the food sufficient, this water is thrown away before the meal is taken, or the ackees are boiled separately and the fruit taken out and mixed with the other ingredients—yam, banana, pumpkin, etc.—while the water is cast aside.

Whatever the nature of the toxin, it seems to be rendered inert, partially or completely, by stimulants in some cases. Patients who were seen in quite an early stage, the initial vomiting period, which, in my opinion, is gastric in origin, had the best chance of recovery, the stimulant—rum, ether, whiskey, brandy—being followed by recovery, as in cases 5, 17, 29.

On the other hand, when the secondary vomiting has made its appearance, which I believe to be cerebral (see next section), such stimulation seems to have no beneficial effect at all. However mild the case seems to be, and however good the general condition, I have never yet seen recovery take place in vomiting sickness if convulsions occur, or if consciousness is once lost, and I have notes now of nearly three hundred cases.

So extensive an outbreak as this in the Montego Bay district is

sufficient to make out a strong case inculpating the ackee, since so large a number of patients being attacked in such rapid sequence, giving almost identical histories as regards the question of diet, cannot be ascribed to mere coincidence.

I, therefore, next undertook experimental work in order to test this, and establish whether any connection exists between vomiting sickness and ackee poisoning. This is described in Section VI.

IV. SEMEIOLOGICAL

The description of the symptoms of this disease also has a history. When several different affections were included under the comprehensive term 'vomiting sickness,' certain symptoms were mentioned which are not really part of the condition as we know it now.

No advantage would be gained by recording here the earlier accounts of the symptoms when, as has been shown in Section I, so many conditions were placed under the head of vomiting sickness.

Potter's report, though mentioning a symptom here and there when speaking of individual cases, does not give any general or concise statement of the symptoms usually present.

The first detailed description was given in my report to the Secretary of State in March, 1913 (Scott, 1913). This need not be quoted here in full, as it was later amplified for a paper read in July, 1914, before the British Medical Association at their Annual Meeting. This will be given subsequently.

Seidelin (1913) described the symptomatology at length.

In my paper referred to above (Scott, 1914), I have stated:—

'The following is a brief description of the usual train of symptoms; the patient, almost invariably a child, goes to bed apparently in ordinary health. In a few instances only there may be a history of slight indisposition, a cold in the head, or some loss of appetite, or a tendency to lie down during the day preceding the actual onset. During the night the child wakes up and vomits—perhaps only once, perhaps three or four times—and complains of "feeling ill." After an hour or so he drops off to sleep again, and some three or four hours later again wakens up, complains of pain in the stomach (pain is used as a term for mere discomfort, frequently among the native population), and almost immediately begins again to vomit, usually frothy mucus, occasionally bile-stained, and later, only watery fluid, with, in most instances, little or no effort, unless the stomach is quite empty, when troublesome retching ensues; if, however, food is taken, either solid or liquid, there is, apparently, effortless vomiting.'

'In a very short time, often a matter of a few minutes, convulsions come

on, and there is "stiffness of limbs" and a "drawing back of the head" (as the parents describe it); coma rapidly succeeds and terminates in death. In some there is no stiffness or retraction, but a general limp condition. The total duration is short, the average being about 12 hours, or a little more or less. The most rapid in my experience was 35 minutes. Frequently, therefore, the patient is not seen during life, and the history is both incomplete and unreliable, as it is obtained by questioning the parents, who, in the general perturbation of sudden and fatal illness, have not noticed particular symptoms, and, unless volunteered by them, their statements, either in affirmation or denial of a definite symptom, are of little value, a reply to a leading question often varying with the form of that question.'

'When seen during life the child is usually in the convulsive or comatose stage; the temperature is rarely high, usually 101-102° F., but it may be normal. The pulse-rate is between 90 and 100, often fairly strong; respirations 26 to 30, regular till towards the end, when the Cheyne-Stokes type may appear. Kernig's sign is present in some of the cases, and may be distinctly more marked in one leg than the other; rigidity of the neck muscles is more common than retraction of the head, and this rigidity is often overlooked because the flexion is not attempted in the strictly middle line. Rigidity may be fairly marked, but when the flexion is combined with lateral movement (as is the case where the test is applied with the child lying on its side) the stiffness may be masked, since lateral movement may be comparatively free in spite of distinct rigidity of the neck muscles. The pupils are usually equal, moderately dilated, and, if the coma is not deep, react normally. In a few there is photophobia, and in those retaining consciousness, general irritability and complaints of headache—not always by any means severe—usually frontal, sometimes general. Delirium is, so far as I have seen, quite uncommon; shortly before passing into the comatose state the child may remark it "feels very bad," but does not call attention to any particular symptom, or locate the pain, if complained of, to any particular spot. In cases which do not end fatally, the state of coma is rarely present; there are vomiting, headache, convulsions, with only temporary loss of consciousness, and recovery is almost as rapid as the onset. Within 24 hours a child who has been seriously ill may be sitting up in bed, and in another 24-48 hours is up and about, showing practically no symptoms, except a little pallor, general debility, which soon clears up, and some residual headache of no great severity; while others in the family, who did not seem to be any worse at the time, have passed into a state of coma, and died in a few hours.'

Being struck by certain outstanding differences, I added the following remarks to the above:—

'I think that from a perusal of the above one is justified in saying that we have at least two classes of cases. In one there is a rise of temperature, rigidity of neck, possibly retraction, Kernig's sign, and generally rigidity with tenderness; in the other the temperature is normal or hardly raised at all, there is general limpness of muscles, no retraction, no Kernig's sign, no general rigidity, and, so far as my own experience goes, it has only been in the former (and not in all of these) that a diplococcus has been seen in and isolated from the cerebro-spinal fluid, never from the latter.'

It will be well to consider in greater detail some of the individual symptoms.

Up to the present time I have notes and records of 265 cases reported to me as having suffered from vomiting sickness. These comprise both the classes just mentioned, those with 'meningitis-like' symptoms and those without.

Those cases which showed symptoms of cerebro-spinal meningitis, or the most prominent of them, such as headache, vomiting, retraction of the head and neck, rigidity of muscles, Kernig's sign, and from whose spinal fluid a gram-negative diplococcus was obtained, have amounted to fifty-six in number.

The diplococcus in several instances gave the typical morphological characters and the cultural and sugar reactions of the meningococcus; in some cases, however, as will be seen in Section IV, certain differences were apparent. Putting this bacteriological differentiation question aside for the moment, and looking at the matter merely from the symptomatological aspect, we are justified in saying that the symptoms and course of such cases differ from those of the ordinary vomiting sickness patients and may be put in a separate category. They have, it is true, both been included under the same heading for some years past, and this has largely helped in keeping the condition obscure. The differences may be shown in tabular form:—

	Ordinary Vomiting Sickness		*Meningitis-like cases*
1.	No prodromata.	1.	May be catarrhal prodromata.
2.	No headache.	2.	Headache present.
3.	Pain not common: when present it is abdominal.	3.	In addition to headache there may be general tenderness.
4.	General limp condition (between convulsions).	4.	Rigidity often.
5.	No retraction of head.	5.	Head retracted.
6.	No Kernig's sign.	6.	Kernig's sign present.
7.	No recovery if consciousness once lost.	7.	Consciousness returning between fits, and even retained during fits if not severe.
8.	Rapid course—few hours.	8.	Course often more prolonged, though sometimes rapid.
9.	Recovery complete, if not fatal in 24 hours or so.	9.	Recovery slower.
10.	Little or no rise of temperature.	10.	Fever present.
11.	Spinal fluid usually sterile.	11.	Organisms present.

In dealing with the symptoms of true vomiting sickness, these cases with meningeal symptoms should be excluded. They are obviously a type of cerebro-spinal meningitis, or, one ought,

perhaps, rather to say a variety of this disease, as a meningococcus-like organism is found in association with the above symptoms.

Of the remaining 209 cases there are 21 which will require a little consideration as to whether they should be classed as ordinary vomiting sickness cases or as 'meningitis' cases.

Case 1. D.H., male, 3 years. History incomplete; obtained from parents after child's death. Spinal fluid was turbid and gave a growth of meningococcus-like organisms mixed with a few coliform bacilli. This tends, therefore, more to the 'meningitis' group.

Case 2. U.W., female, 7 years. History incomplete, 'hiccough and convulsions, with retraction of the head; no vomiting.' Gram-negative diplococci isolated from the spinal fluid, and there were macroscopic signs of meningitis. This fact, coupled with the absence of vomiting, removes it from the true vomiting sickness group and warrants it being included in the other category.

Case 3. E.G., female, 10 years. No details of history, but post-mortem there was a pearly haziness over the whole surface of the meninges, and an organism giving the morphological and cultural characters of the meningococcus was obtained from the spinal fluid, which flowed freely on making a lumbar puncture. Therefore placed under the second (meningitis) heading.

Case 4. W.W., male, 2 years 11 months. History very incomplete, but the spinal fluid was turbid, and yielded a growth on nasgar of a Gram-negative diplococcus. This organism gave the reactions of meningococcus, but also rendered mannite acid. Included under group II.

Case 5. C.W., female, 2½ years. Vomiting, convulsions and coma; Kernig's sign present. Spinal fluid yielded a diplococcus resembling the last.

Case 6. P.B., female, 4 years. Included under meningitis series because there were (in addition to the vomiting, convulsions and coma) retraction of the head and rigidity of the limbs, and, post mortem, macroscopic appearances of meningitis, but the case could not be absolutely proved because no specimens were sent to me from this case.

Case 7. I.S., female, 2 years. Similar to the last: no specimens sent, but macroscopically there were signs of meningitis post mortem, and during life there had been fever, photophobia, retraction of the neck, Kernig's sign, and general rigidity of the muscles.

Case 8. C.B., male, 4 years. Similar history and post mortem changes; no specimens sent.

Case 9. J.A., male, 9 years. Suffered from headache and vomiting; retraction of neck and Kernig's sign present; fever. This patient was given injections of meningococcus vaccine and recovered.

Case 10. M.C., female, 3 years 4 months. Exhibited meningitic symptoms—vomiting, rigidity, retraction of the head, etc.—but not proved, because no specimens were sent. The medical man in attendance stated that he was unable to obtain any fluid by lumbar puncture.

Case 11. E.M., female, 3 years. This is one of the doubtful cases. The history obtainable was not very detailed or reliable. No doctor saw the child until within two hours of death, when it was comatose. There was a history of a cold in the head preceding the onset (? premeningeal catarrh); there were

vomiting, convulsions and coma, but not rigidity nor Kernig's sign when seen by the medical man shortly before death. The spinal fluid, however, yielded a pure growth of the meningococcus. Of such a case, Seidelin states (p. 413), 'Cases of this nature, observed during an epidemic of cerebro-spinal meningitis, would be accepted under this diagnosis without much discussion.' Hence I would include it under heading II.

Case 12. H.S., female, 3½ years. No history of any sort obtained, the child not being seen till after death. Fluid obtained by lumbar puncture gave a growth of a Gram-negative diplococcus which rendered glucose, maltose, and galactose acid (the last very slightly).

Case 13. J.G., male, 8 years. Similar to the last.

Case 14. V.L.C., female, 4 years. No specimens were sent from this patient, but the medical man who attended, stated 'I regarded this as a perfectly typical case of epidemic cerebro-spinal meningitis, with all the classic symptoms . . . An order was given for burial, so I was unable to perform the post-mortem.'

Case 15. D.D., male, 11 years. This is almost certainly a case of meningitis, but, unfortunately, the culture tubes were all broken up in the post. But the history practically suffices to establish the diagnosis. 'There were frontal headache, chilliness; photophobia, 24 hours later; neck and head retracted, irritability with frequent (effortless) vomiting, convulsions and coma.' Total duration of illness 3 days.

The following six, however, though differing from the usual case of vomiting sickness, would be included under that head rather than under the other, i.e. as a possible variety of vomiting sickness.

Case 1. B.D., female, 4 years. This is put into a class different from those just considered, because the history was not that of true cerebro-spinal meningitis, and the coccus obtained was atypical in several respects (see later, Bacteriology, Section V). At the same time it is worthy of note that there was diffuse hyperaemia of the meninges, the brain was oedematous, the spinal fluid flowed freely on making a lumbar puncture. Finally, it was one of the cases of which Seidelin spoke as being accepted as suffering from cerebro-spinal meningitis, had they occurred during an epidemic of this disease.

Nevertheless, it was not a typical case of vomiting sickness, because there was no vomiting.

In placing it, one is in doubt whether to regard it as an atypical one of vomiting sickness associated with a peculiar organism (*M. jamaicensis*), or as an atypical case of meningitis associated with an atypical diplococcus.

Case 2. F.P., female, 3 years. The history of this patient was not very complete; all that could be obtained was that she was suddenly attacked with vomiting, soon succeeded by convulsions, which recurred at frequent intervals till death; and, that the head was 'drawn back'; the spinal fluid flowed freely, and yielded a growth of Gram-negative diplococci, associated with some diphtheroids. The diplococcus had a slight action on glucose only, the rest of the sugars were unaltered.

In this instance, the only differences from typical vomiting sickness were, firstly, the 'drawing back' of the head, and that was not seen by a medical man, but was stated in answer to a question put to the parents after the death of the child; secondly, the organism found. The diplococcus partook more of the nature of the *Micrococcus catarrhalis*, and, both it and the diphtheroid may have

been contaminations, as the cultures were made post mortem, and in the bush, a full twelve hours after death.

Case 3. M.B., female, 4 years. The history of this patient differs from that of ordinary vomiting sickness. Thus, the fits preceded any vomiting by a considerable interval: there is said to have been retraction of the head, and the vomiting is stated to have continued up to the time of death. The spinal fluid flowed freely when a lumbar puncture was made, and smears from this showed well-marked Gram-negative diplococci, but no growth was obtained on the media used. The presence of these in the smears of the fluid, together with the above history, would be sufficient in an epidemic to class the case as one of cerebro-spinal meningitis; but, as the history was not typical, and, as no growth was obtained, this cannot be said to have been proved, and the case has, therefore, not been included in the meningitic category.

Case 4. E.A., female, 3 years. This cannot be regarded as a case of true vomiting sickness. Firstly, there was no vomiting at all, but convulsions with 'trismus, opisthotonus, and retraction of the head' (medical officer's statement). The meninges were found to be intensely congested, with dulling at the base; the fluid was not in excess. No specimens were sent, so the nature of the case cannot be further elucidated, and I fail to see why it was reported as one of vomiting sickness at all.

Case 5. J.E., female, 6 years. Except for a longer duration of illness this patient showed symptoms similar to the last; she lived in the same district. There was no vomiting, and no specimens were sent from the autopsy. The description of the latter was also very meagre, and no positive diagnosis can be arrived at. With the exception of convulsions, none of the usual symptoms of vomiting sickness were present.

Case 6. C.G., male, 6 years. The history of this case was typical of that of vomiting sickness, but, at the autopsy, it was noticed that there was intense meningeal congestion, the cerebro-spinal fluid was in excess, was uniformly turbid, and there were deposits of lymph on the meningeal surface. Lastly, from the spinal fluid was grown a Gram-negative diplococcus giving acid in glucose, maltose, and galactose. This patient was not seen by a doctor during life; the history may, therefore, be faulty, and the case be one of ordinary cerebro-spinal meningitis, but it has not been included amongst those on account of there being no support for it in the history of symptoms.

Having then briefly considered atypical cases, we may pass on to deal with true vomiting sickness. The characteristic symptoms are: vomiting, convulsions, and coma, with general limpness of muscles, and usually early death. As a rule there is no rise of temperature. In a mild case, or rather in a non-fatal case, there are neither convulsions nor coma, and in a few instances the vomiting (the initial vomiting, at any rate) is absent. To the above symptoms may be added one which is occasionally present, namely pain.

Let us consider the symptoms individually as evidenced in 188 cases of true vomiting sickness.

1. *Pain.* This is not a frequent symptom. The word is often used for mere discomfort, and on no occasion to my knowledge was

acute pain present. The patient was never 'doubled up' with pain, nor was it, as a rule, severe enough to cause the patient to resist or resent palpation.

I can find mention of it in only 41 instances out of the 188. In 33 of these the site of the pain was abdominal or rather epigastric, and this symptom when present always appears to have preceded any others.

In the remaining eight the pain was referred to the head, but in them it appeared subsequently to the vomiting, and was either due to congestion in the secondary (cerebral) stage, or to the strain of vomiting, since to this part was referred the pain in the case of four patients who recovered, and in none of them was it severe.

2. *Vomiting.* Seidelin states (p. 452) that out of 40 cases vomiting occurred in 24; it was absent in 6, whereas in the remaining 10 no definite information was obtained.

In my series of 188 this symptom was in evidence in 162, was absent in 15, and no information was given in the histories of 11 patients. Leaving out these last, we may say that it was present in 162 cases out of 177 of which histories were obtained, that is in over 90 per cent.

(*a*) The nature of the material vomited was usually at first food, or, if a considerable interval had elapsed since the last meal, then firstly watery matter, later bile-stained. At times the vomiting may be replaced by troublesome retching. If, as is not very common, the vomiting or retching is severe or prolonged, there may be specks of blood in the vomitus. I, personally, have never seen 'black vomit,' though one can readily understand that, since the congestion may lead to the appearance of specks of blood in the vomit, if this is retained, there may be dark specks in it; this has been described, and I have occasionally seen it myself, but never to such an extent as to render the vomit black.

(*b*) The times at which the vomiting occurs.

In a typical case vomiting takes place at the onset, in fact it may be the very first objective symptom. It is accompanied, as a rule, by considerable effort, and is repeated, it may be two or three times, at short intervals. This is what I term the 'Initial vomiting,' and gives one very distinctly the idea of an effort on the part of the stomach to rid itself of some noxious material.

In cases which recover, this, with possibly abdominal discomfort

(rarely actual pain), is the only symptom. If the effort is prolonged, there may be a little giddiness and headache, such as would ordinarily be produced by vomiting associated with straining.

In cases which terminate fatally, however, after an interval of calm during which there are practically no symptoms, a return of the vomiting occurs, and this time it is of a different character. It is, to a great extent, effortless, and may not be accompanied by any nausea. This vomiting I designate the 'Secondary vomiting,' and it is, in my opinion, cerebral in origin, owing to its character and also because it is usually followed almost at once by other nervous symptoms—convulsions and coma.

Turning to my series of cases, in consideration of the fact that, as already stated, patients who recover do not exhibit this secondary vomiting, we may say that out of the 148 fatal cases in which the vomiting is mentioned, 86 showed this symptom both at the beginning and later before the onset of other cerebral symptoms.

At the same time one or other may be absent. Thus, the initial vomiting only is seen in cases which recover, and the patients never reach the stage when the secondary, cerebral symptoms appear; again, this initial vomiting is, as it were, suppressed in the very rapid and acute cases. There is an attack of vomiting which is so rapidly followed as to be almost accompanied by the convulsions and coma, the entire symptoms being cerebral, owing to rapid absorption of the toxin from an empty stomach, and death may then take place in an hour or even less.

In much rarer instances the secondary attack of vomiting is suppressed; the patient may pass through the initial attack and appear to improve; then, after a considerable (but varying) interval, he is seized with convulsions, passes into the comatose stage and dies.

Lastly, vomiting may be absent; the cerebral symptoms may be the first indications of anything wrong. For example, a child of four years of age was quite well when she ate her dinner at 1 p.m. Two hours later she felt out of sorts and went to lie down. An hour or so afterwards her mother went to awaken her, but could only partially do so; twitching of limbs and slight convulsions came on, and the child lost consciousness altogether and remained comatose till death.

Such cases have been diagnosed, somewhat paradoxically, as 'Vomiting sickness without vomiting,' and I am of opinion that they do occur, though exceedingly rarely. Such a diagnosis, however, could not be made unless (i) true vomiting sickness cases were occurring at the time, (ii) all other causes could be excluded, or (iii) the post-mortem signs, especially microscopical, were those of vomiting sickness (see next section).

The vomiting sickness returns are, I regret to say, unduly swelled during the season at which the disease prevails by reports of deaths as such merely because the child had an attack of vomiting sometime during its final illness, or if death occurs and no adequate cause is found, the case is certified, as already stated, as one of 'vomiting sickness without vomiting,' and the autopsy is thus avoided.

Briefly, to sum up with regard to this symptom:—Initial vomiting only was present in 65 cases, including 34 who recovered; secondary vomiting only in 11; while both initial and secondary occurred in 86 instances.

3. *Convulsions.* Opinions as regards this symptom are a little varied. In some histories where this symptom is mentioned, further enquiry elicited the fact that merely slight twitching movements of the limbs were noticed. In other cases there were definite tonic contractions of muscles, lasting for a few seconds only, while again some were described as clonic. The uncertainty arose often from the fact that the patient was not seen by a medical man during life, and the statements of parents, other relatives, or friends had to be relied on.

Looking over my notes: In the cases which recovered, 'slight twitching' movements occurred in one patient only, a child of four years of age. Everyone has seen slight twitching movements in a child asleep, apparently in ordinary health, or with a little dyspeptic disorder, so I think one may safely say that in no cases which recover are convulsions seen.

In Seidelin's table the only case of recovery in which convulsions are stated to have been present is noted as not being a definite case of vomiting sickness. His observations in this respect, therefore, agree with my own.

In 151 fatal cases convulsions were definitely stated to have been present in 101. But of the 151 no mention, either positive or

negative, as regards this symptom, is made in 24 instances, and in 8 more the point is doubtful, the history being conflicting. Deducting these, we may say that of 119 cases where convulsions are definitely spoken of, they were present in 101, or 84·87 per cent., and absent in the remaining 18, or 15·13 per cent.

4. *Coma.* In 33 instances no mention is made of this, but in all the other fatal cases it was present, and in none of those who recovered.

Seidelin puts a + under the column of 'coma' in three cases which recovered, but from his description of the patients they could hardly be said to be comatose. Thus, the first case was a girl of four years of age, who showed 'slight collapse' after two hours' vomiting; the second, a baby aged two months (eight months is stated in the table, two in the detailed report) who suffered with 'continuous vomiting and collapse', while the third was a boy of twelve years who, when seen after 'vomiting and retching' for many hours, appeared 'weak and drowsy.'

The coma in most of the cases which I have seen was deep; as a rule there was absolute unconsciousness with no conjunctival reflex, though in one or two there was, at an earlier stage of the coma, some irritability when attempts were made to rouse the patient, but this 'cerebral irritation stage' was transitory and soon passed into one of deep coma.

Other nervous symptoms—Kernig's sign, rigidity of neck muscles, rigidity of muscles generally. Apart from 'meningitis' cases, these were rare in patients seen by a medical man during life. Kernig's sign is only mentioned twice, and in one of these the patient recovered and the symptom had disappeared within twenty-four hours of its appearance, which is sufficient to cast some doubt as to its reality. The second was atypical in many respects; in fact, I have a note to the effect that I cannot understand the reason for the diagnosis, except that the patient, a child of five years, died during the vomiting sickness season. It has been included because the case was so reported and has been so entered in the records. The history of symptoms was very meagre: 'sudden onset, convulsions, no vomiting, Kernig's sign present, teeth clenched, temperature 99·6° F.' There is no other statement and no specimens were sent from this case. May not the so-called Kernig's sign noted

here have been part of the general rigidity associated with the 'clenching of teeth and convulsions'?

In three instances only is 'rigidity of neck' stated to have been present, and then only 'during the fits'; there was no rigidity noted in the intervals.

The same remark applies to four out of the five cases in which rigidity of other muscles is mentioned. In the remaining instance the patient was not seen during life, but, on questioning the mother after the child had died, the history given was that 'the mother woke up to find her child unconscious with teeth locked, arms flexed and stiff, hands clenched, legs stiff, and head drawn back. In other words, the child was in a tonic convulsion.

5. *Fever.* This is not a common symptom at all. The temperature in the majority of cases is normal, and a rise above 101° is quite unusual. In one case 102·4° is mentioned, and in two others 101°; with these exceptions the highest recorded is 99° F. We may take it, therefore, that in ordinary cases of vomiting sickness there is no fever.

This practically disposes of all the symptoms; the pupils are neither unduly dilated nor contracted, and are equal in all which I have seen, and in all those in which the state of the pupils has been noted by others.

Finally, a few remarks on the questions of age, sex, and duration of illness.

Age. A perusal of Tables I and IV appended will show that the disease is to a great extent one of childhood. Babies in arms are not attacked; only two cases occurred under the age of one year. 87, or 44·84 per cent., however, occurred in the first quinquennium; 58, or 29·89 per cent., during the second; 20, or 10·31 per cent., in the third; and only 2, or 1·03 per cent., in the fourth.

The mortality rate is high in all these periods. Thus, of those attacked between the ages of one and five years 85·06 per cent. died; between five and ten years 86·21 per cent.; in the third quinquennium, out of the 20 attacked 15 died (75 per cent.).

Sex. The disease shows practically no predilection for sex amongst those at the susceptible age. Thus, of 145 cases occurring in the first decade of life (74·74 per cent. of the whole) 65, or 44·82 per cent., were males, and 55·18 per cent. females, and the death-rate

is closely similar, namely, 45·96 per cent males and 54·04 per cent. females.

Dividing the ten years into two quinquennial periods, we see that of 87 cases occurring up to the age of five years 35, or 40·23 per cent., were males, and 52, or 59·77 per cent., were females. Of fatal cases during the same period 43·24 per cent. were males and 56·76 per cent. females. Between the ages of five and ten years 58 cases occurred, of which 30 were males and 28 females; 50 died, of whom exactly half were males and half females.

Duration of Illness. In 140 instances the duration of illness was given. The shortest recorded was in a female child of one year, when death took place within half an hour. The average duration of the whole 140 works out at 12·72 hours. Sex has no influence on duration, for, although of those whose duration is given 82 were females and 58 males, the length of illness between time of onset and death (including, when present, the period of calm) works out at 12·5 hours in the case of males and at 12·89 hours in females, a difference of only 23 minutes.

This section, dealing with the symptomatology of the affection may best be summed up by giving a brief description of four different cases: —

1. Symptoms in a mild case.

A girl, P.M., 9 years of age, was given the soup obtained from fish, bananas and ackees boiled together, at noon, on February 28th. Prior to this she was in her usual good health, and nothing was noticed until, at 2 p.m., she complained of abdominal pain, and suddenly vomited. This continued at intervals for three hours, and the child then went to bed and slept well. Early the following morning she again complained of pain, and vomited. A doctor saw her, but, except for slight epigastric pain, a furred tongue, and a subnormal temperature (97·6° F.), there were no abnormal physical signs present. The pain was not severe, since palpation was not resisted. She was given some stimulant mixture containing ether and ammonia. No further vomiting occurred, and recovery was complete by the same evening.

2. Symptoms in a case apparently mild at first but terminating fatally.

P.H., male, aged 3 years. In usual good health until the evening of March 5th, when he was given a meal of vegetable soup. About two hours later he suddenly vomited, although he had made no complaint of pain. He rapidly recovered and seemed quite well on going to bed an hour or so afterwards. He slept well till just before dawn, 6th, when, without any warning, he again vomited, and was very shortly afterwards seized with convulsions; coma supervened, and death took place at 11 a.m.

The total duration from the first onset of vomiting was 16 hours; there was a symptomless intermission of 8 to 10 hours, and death occurred in five hours after the onset of the secondary vomiting.

3. An average case. This corresponds very closely to the last but the interval is one of improvement, not always of absolute cessation of symptoms.

A girl of 6 years of age went to bed at the usual time, apparently in perfect health. Early the next morning, without complaining, she suddenly vomited yellowish, watery matter, and, in the course of the next hour, vomited twice. During the day she felt sick and stayed in the house. She made no definite complaints and took her food when it was brought to her, but was clearly not quite herself. Towards evening she improved and went to bed, and slept well during the night. The next morning, about 6 a.m., without any obvious cause, and without any effort, she vomited frothy and watery material, and, within a few minutes was seized with convulsions and passed into a state of coma. She did not rally at all, and died at 2 p.m.

4. A case in which convulsions were absent.

A child of 12 years of age, with no record of any previous illness, left home in her ordinary health for school, about three miles away. About midday she had a meal, the nature of which was uncertain. She made no complaint until between 3 and 4 p.m., when she stated that she had pains in her stomach and began to vomit. She vomited three times and felt much better. She then started for home. On the way she felt ill and rested now and again, vomiting at intervals. She did not reach home till 7 o'clock. Shortly afterwards, without any convulsions being observed, she passed comparatively rapidly into a state of coma, which deepened till death, some five hours later.

A case of so-called 'Vomiting sickness without vomiting' has already been described (p. 22).

A reasonable interpretation of the symptoms is that some poison is taken, or some substance which acts as a poison after entering the stomach. If the initial vomiting is sufficient to get rid of this substance, no further symptoms occur and recovery rapidly ensues.

If this is not the case, there is an interval—a more or less quiescent period—during which absorption is going on, and then follow symptoms due to the action of the toxin on the higher centres—secondary (cerebral) vomiting, twitching and convulsions, drowsiness and somnolence deepening to coma and death. In rarer instances it is possible that the cerebral symptoms (convulsions, drowsiness, coma) are the first noticeable; there is no preceding vomiting—the so-called 'vomiting sickness without vomiting.'

V. PATHOLOGICAL

I. Anatomical

Prior to the time of Dr. Seidelin's investigation (1913-1914) there had been no systematic detailed description of the morbid anatomy of this affection. Potter's report (1911) gives brief notes of some twenty autopsies, but these comprise little beyond macroscopic appearances.

Seidelin sums up the anatomical aspect of the question briefly thus (p. 394):—'The most constant and most remarkable pathological changes were: fatty metamorphosis of liver, kidneys, and other organs; necrobiotic changes of epithelia in pancreas, kidneys, and liver; swelling and hyperaemia of lymph nodules; hyperaemia of many organs, including the meninges, and a tendency to haemorrhages; widespread oedema of the connective tissues.'

His descriptions of the various organs and tissues are excellent and full of detail, and in this present section I quote largely from his report, as presenting the question in a clear and adequate manner which can hardly be improved upon.

A. *Macroscopic*

(1) *General condition.* There is a popular idea that the disease occurs mostly in emaciated and badly nourished subjects, if not actually confined to such. This is quite erroneous. Seidelin notes 10 out of his 62 cases as emaciated, and one of these was not definitely a case of vomiting sickness, at least there is a ? mark under the heading 'clinical character.' In my series two were reported as 'emaciated' and three others as 'poorly nourished.' Obviously, therefore, the disease is not one which singles out the weak and debilitated.

(2) *Jaundice.* Seidelin mentions this in his table in one instance only, but he has evidently excluded those with 'slight jaundice of sclerae,' for this is mentioned in the detailed accounts of six cases; in one it is stated to have been general, in three slight, and in two confined to the sclerae.

In my series there was 'slight yellow discoloration of the sclerotics' in seven; in another it was more marked but similarly limited. It must be remembered that the sclerotic of the native often shows an apparent yellow discoloration which on closer examination

proves to be a thin layer of fatty deposit, and might be casually mistaken for jaundice.

On the other hand slight jaundice does occur, and the condition of the liver present in many cases is sufficient cause for this.

(3) As regards other external conditions, some district medical officers have described sores and fissures at the angles of the mouth. I have occasionally seen them in vomiting sickness cases, but so seldom that I am inclined to think that they are accidental, due, perhaps, to syphilis or yaws. Personally, I recollect seeing them but twice.

For purposes of description of the internal organs the contents of the head, thorax, and abdomen will be considered in order.

Brain and Spinal Cord and their Meninges

In almost all cases fluid is obtained fairly readily on a lumbar puncture being made. As a rule, the spinal fluid is clear and flows drop by drop; occasionally it has been cloudy, and it may flow in a steady stream as if under considerable pressure.

The meninges of the spinal cord are often hyperaemic, in fact usually so. The cord itself may in some instances show hyperaemia, but quite as often nothing abnormal is detected.

The cerebral dura is in the majority of cases normal in appearance, but may be hyperaemic; the pia mater on the other hand is more often congested, and the vessels may be engorged, especially on the convex surface of the brain.

There is no fibrinous exudation, though a certain degree of serous meningeal exudate is not uncommon; the brain surface may or may not be congested, and the substance also. In some cases, particularly if the convulsions have been severe or prolonged, minute petechial points are seen on section of the brain substance.

The ventricles are not distended, and apparently the fluid is never markedly in excess.

The hypophysis cerebri may share in the general hyperaemia.

Thoracic contents:

The *Thymus* in one of my cases appeared enlarged, but in none of the others was any abnormality seen.

The *Pericardium* often contains a few cubic centimetres of pale, clear fluid, but not in excess.

As regards the *Heart*, in many cases no abnormality is apparent; it is not uncommon, however, to see hyperaemia of the epicardium, sometimes petechiae and even small ecchymoses. These, when present, are more often visible on the ventricular surface near the auriculo-ventricular junction and posteriorly. They are very rarely seen on the interior aspect of the heart muscle.

The myocardium usually appears normal, but at times there are pale, greyish or yellowish streaks, or patches of small extent. In such the consistence may be a little diminished.

The *Thyroid* is normal in appearance.

The *Pleurae* may show a few petechiae and even minute ecchymoses, but in the majority of cases nothing abnormal is seen. Occasionally a few cubic centimetres of straw-coloured fluid are present in the pleural cavities.

The *Larynx* in some is hyperaemic, as is also the trachea. The trachea and bronchi may contain some frothy mucoid secretion, which in a few instances is blood-stained.

The mucous membrane of the bronchioles may show a diffuse catarrhal condition, a frothy mucus escaping on section. Hypostatic congestion of the bases of the lungs is often present, and these organs may appear hyperaemic and oedematous. By no means infrequently, definite small haemorrhagic infarcts are seen scattered in various parts of the lungs.

The bronchial lymph glands may be hyperaemic and slightly enlarged.

Abdominal contents:

The *Peritoneum* in all cases appears normal, there is no sign of inflammation, and no fluid present in the peritoneal cavity.

The *Stomach*. In some cases nothing abnormal is noticed, but in the majority the mucous membrane is congested, especially over the posterior wall and along the lesser curvature; at times there are several petechiae seen, and occasionally small ecchymoses. The contents are usually grumous, mucoid, frothy material; occasionally dark specks are present.

The *Duodenum* presents the same characters as the stomach. If the latter is normal the former as a rule is also; if the stomach is congested a similar hyperaemic state of the duodenal mucous membrane is also found in most instances.

The *Intestines*. Ascarides are present in some cases, and there may be patches of hyperaemia, especially if the worms are numerous. Otherwise the intestinal mucous membrane is apparently not affected.

Spleen. The capsule is smooth and transparent, the consistence of the organ is usually normal, but may be diminished. Petechiae are occasionally visible on the surface, and the follicles may appear prominent on section.

Liver. Capsule smooth and transparent. The colour of the viscus varies; it may be dark purple, or at times is of a reddish-grey colour with pale patches; again, in a few cases it may be uniformly yellowish-grey. The consistence in such is diminished.

Kidneys. Capsule normal and easily detached; the stellate veins may show out well. The surface may be dark and show capillary hyperaemia; in other cases the surface is pale, and dilated stellate veins are prominent. On section, venous and capillary hyperaemia may be seen, but by no means invariably. The cortex and bases of the pyramids may be hyperaemic, while the apices of the latter are pale. There may be minute haemorrhages and yellowish stripes in the cortex, and signs of a diffuse parenchymatous nephritis.

The suprarenals are in a few cases hyperaemic, but usually there is nothing abnormal detected in them.

The *Pancreas* in many instances appears normal, but may be hyperaemic and of diminished consistence.

In four cases reported as vomiting sickness I have met with an acute haemorrhagic condition of this organ.

The mesenteric glands are usually enlarged, and in fully half the cases they are found, on section, to be distinctly hyperaemic.

B. *Microscopical*

The spinal fluid is in most cases normal. In some, after centrifugalisation, the cells contained are seen to be mostly mononuclears; a few polymorphonuclears may be present, and in some cases (the 'meningitic' type, rarely in the true vomiting sickness type) Gram-negative diplococci.

Brain and Spinal Cord. The former often shows a condition of hyperaemia, the latter less commonly. The pia mater may show some oedema and patches of microcellular infiltration.

Heart. The muscle-fibres may show fragmentation, and more rarely minute fat droplets.

Lungs. In parts of the lung tissue taken from the bases there is general congestion; the bronchioles exhibit swelling of the cells of the mucous lining, and shedding of epithelium, the alveoli also contain shed cells. In the infarcted parts the alveoli are filled with red blood corpuscles, leucocytes, and shed epithelium, but no organisms are discoverable.

In a few cases fat droplets have been seen in the epithelial cells.

Stomach. In the majority of cases there is localised hyperaemia of the mucosa, and there may be diffuse microcellular infiltration of the mucosa and submucosa. Less often minute haemorrhages are visible.

The Duodenum exhibits similar changes, but less frequently. There may be hyperplasia of the lymphoid follicles with slight microcellular infiltration and patches of hyperaemia. The glands of Brunner are at times prominent.

Liver. In most there is irregularly distributed capillary hyperaemia, and there may be microcellular infiltration of the periportal tissue. In the majority of cases the cells of the parenchyma show a fatty change, which varies between the very slight and the intense. In some cases this fatty change appears to be more marked in the neighbourhood of the portal vessels, in others it is fairly equally distributed over all zones of the lobule.

Spleen. In many cases there is little or nothing abnormal to be made out. There may be diffuse hyperaemia and the follicles may show necrobiotic changes. Both these conditions may be of irregular distribution.

Kidneys. In most instances there is renal affection. Hyperaemia of varied degree is usually found; this may, in parts, be intense and small extravasations occur. The stroma in some appears to be oedematous and even a little increased, and there is slight microcellular infiltration. The glomeruli may be retracted, and in the spaces between them and Bowman's capsule coagulated serum is seen. In other cases the glomeruli may be distended and hyperaemic. The epithelium of the convoluted tubules and of the ascending loops of Henle shows necrobiotic changes in some cases with granularity and vacuolization and fatty changes, especially at the basal portion of the cells. The nuclei may show karyolysis.

The adrenals in a few instances showed considerable hyperaemia, and, rarely, some vacuolization of the cells.

Pancreas. Necrobiosis of the cells, variable in degree and extent, is common; the cell limits in these cases are badly defined and the nuclei stain poorly. Langerhans' islets are usually (but not always) well preserved and definite.

The limits between the well preserved and the necrobiotic cells may be quite sharp, cells of the two types being seen sometimes side by side in the same lobule or even in the same alveolus.

Sometimes irregular infiltrating haemorrhages are seen, and in many cases the epithelial cells contain several small fat-droplets.

Lymph Glands (especially mesenteric) show a diffuse hyperaemia, patches of necrobiosis, and moderate oedema of stroma.

The *Urine* is, in the majority of cases, plentiful. Possibly, as Seidelin thinks, excreted prior to the onset of the final illness. It is usually normal—clear, acid in reaction, without deposit on centrifuging. But in some there is a little albumen, a few granular and hyaline casts, and occasionally some red blood corpuscles. No organisms are seen.

II. Bacteriological

Upon this subject I have very little to say, because investigations which have been carried out during the last two years make me incline more and more to the opinion that the disease has no bacteriology. I mean by this that the organisms which have been found in some of the patients described as suffering from vomiting sickness are not causative.

The bacteriology of the affection is largely of historical interest.

Dr. Seidelin's account on pages 458-465 of his report is excellent, and well worth studying by any who wish to trace the stages through which the descriptions of this disease have passed.

As I have stated in the previous section, true vomiting sickness cases can usually be differentiated clinically from the 'meningitis-like' ones in which the diplococci have usually been found. In the season of 1913-14 out of twenty cases I obtained the diplococcus only twice, and in one of these the cultural reactions differed from those of the meningococcus. In another the coccus was atypical in tending to grow in chains and also in producing acid in mannite and raffinose.

In one case only amongst those seen by me in the Montego Bay outbreak this year were diplococci seen in the spinal fluid, and in this instance no growth was obtained. They were visible only in a smear of the fluid after centrifugalisation; cultural attempts failed completely.

My opinion, the result of prolonged investigations, is this:— If we set aside true meningitis cases in which the meningococcus is found, such as the 'Peart' series in 1912, which are few; and if we set aside also cases with anomalous symptoms which have been previously included under the comprehensive term of vomiting sickness, and of which I have spoken in the last section; then the organisms found in the remainder—the true vomiting sickness cases proper—are accidental concomitants, or at the most are part causes only; and even of this latter idea I have grave doubts.

My reason for this are three:—

(i) In by far the majority of cases no such organisms are present at all.

(ii) When cultivated and injected in a living state and in large doses into laboratory animals—guinea-pigs, rabbits, Belgian hares—no untoward effects were discovered. Inoculations were made subcutaneously, intraperitoneally, intravenously, and intracardially.

(iii) The organisms showed extensive variations. They varied as regards—(*a*) Form: sometimes occurring in groups, sometimes tending to chain formation; (*b*) Size: sometimes as small as the gonococcus, say, 6μ, at other times more than double this; (*c*) Staining reactions: sometimes readily decolorized by Gram's method, at other times with considerable difficulty; (*d*) Sugar reactions: galactose is sometimes rendered acid, at other times not; lactose, saccharose, mannite, raffinose are in some cases acted upon.

As Seidelin very pertinently remarks (p. 464): 'Experience has 'shown in several diseases that a germ which has for some time been 'regarded as a pathogenic one, has, on close investigation, been 'reduced to a secondary position as representing only a complicating 'infection.'

With respect to vomiting sickness I would fully endorse this, and, perhaps, go even a step further and say that vomiting sickness

pure and simple is not, in my opinion, a bacterial affection, but an intoxication; that the organisms found in the spinal fluid are either accidental and non-pathogenic, or at most may intensify the nervous symptoms by causing an increase in the amount, and consequently in the pressure, of the cerebro-spinal fluid.

The diplococcus is much more often absent than present even in typical cases, and in by far the majority of instances cultural attempts, as well as direct examination of the blood or spinal fluid, yield only negative results.

VI. EXPERIMENTAL

My own work in this connection will be described in detail later in this section. When I started investigating the question of ackee poisoning early in the present year (1915) I was unaware that any work had previously been carried out on the subject. I was informed, however, by Mr. Robert Simmons, F.I.C., the Deputy Island Chemist of Jamaica, that a predecessor of his, Mr. J. J. Bowry, had undertaken an investigation into the nature of the ackee poison as long ago as 1886. Mr. Simmons very kindly looked up the notes of this enquiry and sent me an abstract of them, and also a brief tabular statement of twelve deaths which followed the eating of the fruit of the ackee (*Blighia sapida*).

He also gave me the following letter which probably led to Bowry's investigations being undertaken:—

'Montego Bay,
February 26th, 1886.

'Dear Mr. Bowry,

'I have, with great interest, read your favour of the 25th inst., along with enclosed extract from a medical man, giving particulars of the deaths of several children in Vere.

'After a careful consideration of all the facts I cannot arrive at any other conclusion than the cases there mentioned were those of narcotic poison, and the particular poison in this case, I feel confident, was that of the "Ackee," as the symptoms exactly tallies (*sic*) with my own experience of the poisonous action of this fruit. If you will go to a little further trouble, and have enquiries made, I think you will find that these children had been playing probably the day before under ackee trees, somewhere in the district, the probable inference is that they picked up portions of the decayed fruit and ate it. A great many times this condition of affairs have (*sic*) been brought to my notice, in the majority of the cases I have not been able to prove that they did actually eat; but, the history you always get is that they were playing under the tree, and, more out of mischief, eat small portions of the decayed fruit that had been for some days on the ground.

At a period of about 12 hours after the action of the poison commences, exactly as described in the extract from the letter you sent me, and always ends fatally. In my experience I have never seen a case of recovery, the prominent symptom being gradually increasing coma.

'I will write you again in a few days.

I remain, Dear Sir,

Yours very truly,

ALEX. McCATTY.'

I have quoted the letter verbatim. (H. H. S.)

Bowry's experiments in ackee poisoning were undertaken to discover, as a chemist, the nature of indigenous poisons in Jamaica. There is nothing whatever to indicate that he suspected any connection between ackees and vomiting sickness. This connection dates only from my investigations this year at Montego Bay.

Returning to Bowry's results, Mr. Simmons states: 'He '(i.e. Bowry) ascertained that when the white portion of the ackee 'was soft it was poisonous; that an unripe or decayed ackee was 'poisonous. The edible portion of ackees taken from a broken 'bough was decaying, whether the fruits were open or still closed. 'He found that ackee fruits with no, or very small, or undeveloped 'seeds were those specially prone to develop the poison.'

In Bowry's own words: 'In each case which has come under my 'notice of ackee fruits being poisonous, it has been fruit without well-'formed seeds which have been deadly. Not that fruits with 'abortive seeds are necessarily poisonous; when ripe and perfectly 'fresh, I believe them to be as wholesome as those with perfect seeds, 'indeed by actual experiments I have found such ackees to be 'wholesome. But these seedless fruits are apt to become over-ripe, 'stale, even decayed, without opening.

'It would appear that the decay which takes place in the 'unopened fruit results in rendering it deadly, as decay in open 'fruit does not. When there are well-formed seeds in a fruit it must 'open before decay begins, while seedless fruits will often remain 'closed until the edible portion has become quite soft and poisonous.'

The attached table was compiled from notes of cases of undoubted ackee poisoning with which Bowry dealt, and in which specimens of the ackees supposed to have caused death were forwarded to him and proved by him to be poisonous. He obtained extracts from suspected ackees and proved their toxicity on various animals, but he never succeeded in obtaining the same poisonous extract from the viscera of a person poisoned by ackee.

To quote again Bowry's own words: 'There is no known method 'of recognising poisonous ackees after they have been eaten, even if 'any remain in the stomach after the vomiting which poisonous 'ackees cause, for persistent vomiting is the most marked symptom 'in cases of ackee poisoning.'

He draws attention to the increased difficulty of attempting to recognize the poison in viscera owing to the stomach contents being lost by vomiting. In one case he noted that the fruits from a 'broken bough' were undergoing a peculiar form of decay, the edible portion being soft and slightly discoloured, and the membrane, which is usually red and tough in good fruit, was broken and soft in the shrivelled ackees.

An extract prepared from the edible portion of these fruits proved poisonous to mice and cats, with symptoms resembling those seen in the persons who died. A similarly prepared extract from fresh ackees bought in the market proved harmless.

He found that the edible portion of wholesome ackees, minced and allowed to stand for a week until mouldy and rotten, did *not* yield a poisonous extract, but that some of the same ackees similarly treated for the same time, with the addition of a small quantity of the ackees from the broken bough, yielded a very poisonous extract.

This showed that it was some specific change which led to the production of the poison, and not ordinary decay. He held the view, therefore, that the poison was produced by some enzymic or catalytic action.

As regards tests, he writes: 'No tests other than physiological 'are yet known for the ackee poison, and, as vomiting is a most 'marked symptom, all poison still unabsorbed into the system is 'entirely removed from the stomach before death takes place.'

He also incidentally refers to the difficulty in obtaining reliable information as to symptoms, their nature and time of appearance, and the interval of time elapsing before death.

In commenting on this, he says: 'Immediately the Police take 'a case up the friends and relatives of the deceased become very 'unwilling to give any information, fearing that they may be 'charged with some offence in connection with the occurrence. They 'contradict each other, every item of importance has to be dragged

'from them, and no satisfactory conclusion can be drawn from 'their statements. But for this fear of ulterior consequences many 'cases which now remain shrouded in mystery would become plain.

'I have known the relatives of a deceased child try to make out 'that it could not have eaten ackees, although eventually they 'have admitted there was an ackee tree in the yard from which the 'child could have obtained fruit, and I have found the fruit to be 'of a very suspicious character.'

The previous sections of this paper have shown how frequently I have met with the same difficulty in eliciting true statements on these points from the relatives.

In view of my own findings, Bowry makes two other remarks of interest. The first is that in his experience the history of ackee poisoning showed that children constituted the greater number of victims, 'probably because they are not acquainted with the conditions under which ackees are considered by adults to be dangerous, and so pick them unripe, off broken limbs and from the ground, and eat them with fatal results.' The second is that he is 'disposed to think that many deaths which have been set down to ackee were really due to cerebro-spinal fever, and that it is a mere unconnected coincidence that the climatic influences which give rise to the disease occur during the ackee season.'

When one considers that, in the light of my remarks in previous sections of this paper, I arrived at the same conclusion—that vomiting sickness and cerebro-spinal meningitis cases have been confused and included together in the same category—the statement of Bowry's gains increased interest, and had I known of it, I should have been saved months (even years) of work.

His table, attached, is of interest in that two-thirds of the cases occurred in children within the first decade as compared with 74 per cent. of my series of vomiting sickness patients. The symptoms are similar, namely, vomiting, convulsions, coma and death. The duration of illness he estimates a few hours longer than in my series, but it must be noted that he definitely remarks on the difficulty he found in obtaining reliable information as to the interval of time elapsing before death took place.

The most important defect in the table is the absence of any description of the condition of the organs and tissues,

Age	Symptoms	Death in	Certified cause of death	Post-mortem Results						Remarks
				Brain	Stomach	Stomach contents	Intestines	Liver	Kidneys	
4	Severe vomiting	13 hours	1. Acute irritant poison 2. Gastritis	Intensely congested	Mucous membrane irritated and congested	Frothy, thin, dirty liquid	—	—	—	Deceased and his brother (aged 7) ate an unopened ackee
8	Persistent vomiting	13 hours	Syncope	—	—	—	—	—	—	Vomited up portions of ackees
7	Convulsions	16 hours	1. Ackee poisoning 2. Syncope	Congested	Normal	Thin fluid	—	Swollen	Congested	Ate raw ackees
14	Vomiting, collapse, coma	About 24 hours	1. Ackee, ptomaines ? 2. Syncope, asphyxia	—	Normal	Yellow fluid	Normal	Normal	Normal	Ate freely of ackee soup. The father also partook of some, was very ill afterwards, but recovered
37	,,	,,	,,	—	,,	,,	,,	,,	,,	
2	Coma	15 hours	—	—	—	—	—	—	—	Ate ackees
2	Vomiting, headache, fits	About 42 hours	1. Poison from eating unripe ackee 2. Convulsions	—	—	—	Distended	Enlarged	Congested	All ate from a heap of decayed ackees which had been lying in the yard for about a week
4	,,	,,	,,	—	—	—	,,	,,	,,	
9	,,	,,	,,	—	—	—	,,	,,	,,	
11	,,	,,	,,	—	—	—	,,	,,	,,	
Child	Fits	14 hours	—	Congested	Normal	Thin gruel-like liquid	Normal	—	Congested	Ate uncooked ackees
20	Vomiting and severe purging	72 hours	Ackee poisoning	—	Not inflamed	Thick matter	Distended	Enlarged	Healthy	Picked ackees, cooked them next day, ate them and gave some to her child, aged 4; child became ill, but recovered. Deceased was a weak women

macroscopic or microscopic. The reason for this was, that there was no pathologist in the island until many years later.

Seidelin (1913) mentions briefly (pp. 460-462) some experimental work with some of the diplococci isolated from cases seen by him, but they did not appear to have any pathogenic properties as regards the animals he employed.

From 1886 to the present year (an interval of 29 years), when my investigations into the Montego Bay outbreak, recorded in Section III, led me to suspect ackee poisoning as the cause of the mysterious vomiting sickness, the subject appears to have been completely dropped, in abeyance if not altogether forgotten, except for the two or three casual remarks to which I have called attention when describing the history of the disease, in Section I.*

At my request, Mr. Simmons kindly prepared some ackee extracts made from fruits with undeveloped seeds and (*a*) firm arilli, (*b*) decayed arilli, using absolute alcohol, amyl alcohol, ether, and petroleum ether.

The method of preparation of each was as follows:—

(1) *Ackees with imperfect seeds and firm arilli.*

(A) The macerated arilli and placenta were treated with absolute alcohol in the cold for two days. After filtration, the solvent, which was of an orange colour, was evaporated under reduced pressure at 60° C. To the residue more absolute alcohol was added, and the precipitate formed removed by filtration. The filtrate was evaporated under reduced pressure at room temperature. The residue was dissolved in water, filtered, and tubed.

(B) The arilli and placenta were allowed to stand under reduced pressure for two days, at the end of which time the arilli had become soft and mouldy. The filtered absolute alcohol extract was greenish in colour, and addition of water produced a milky appearance. The weak alcoholic liquid was extracted with amyl alcohol, the latter removed, and the aqueous portion treated with petroleum ether to remove traces of the amyl alcohol. The aqueous portion was then evaporated, taken up with water again, filtered and tubed.

* Section I, dealing with the history of this disease has been abridged; the statements referred to may be seen in the official reports of the Island Medical Department from which the (unabridged) history was compiled.—H. H. S.

(2) *Ackees with undeveloped seeds and decayed arilli*

(C) An absolute alcohol extract made in the cold was gently evaporated, and left a brown resin which was insoluble in hot absolute alcohol, but soluble in water. The resin was removed by filtration, and the liquid concentrated. The residue, dissolved in water, was treated with basic lead acetate, excess of lead removed by hydrogen sulphide, and the filtrate evaporated. The residue obtained was still of a brown colour, and contained minute acicular and cubic crystals. The aqueous solution of this residue was tubed.

(D) The resin above was dissolved in water and tubed.

(E) A hot absolute alcohol extract on cooling deposited minute crystals which re-dissolved on heating. The evaporated extract treated with water resulted in the separation of an oil. The filtrate from this was evaporated to a smaller bulk, and tubed.

(F) An extract of ackees dried *in vacuo* was made with petroleum ether; the evaporated extract left a considerable quantity of yellow oil. The residue was treated with water, the oil removed, and the solution concentrated and treated with absolute alcohol. The precipitated matters were filtered off, the liquid evaporated, taken up with water, and tubed.

(G) Similar to the last, but using ether instead of petroleum ether.

I tested these by intragastric administration to guinea-pigs, rabbits, and Belgian hares. The results, however, were indefinite; but the experiments are mentioned because I think that one can learn something from the very fact of their failing.

In my opinion the failure may be attributed to one or other of three causes:—

(i) The amounts of extracts made were small. Mr. Simmons was very busy at the time, and the preparations had to be carried out in addition to his ordinary routine work, which is heavy.

(ii) The extracts were all made with alcohol or ether as the primary menstruum. Judging from the results of early treatment, if stimulant in the form of alcohol (rum) or ether is given, recovery ensues. This is very probably due to precipitation of the poison rendering it non-absorbable if not inert (see later, Section VII). If this is so, the filtrate from treatment of the ackees with alcohol would very likely not contain the poison.

(iii) The animals employed were all herbivora. I could not at the time obtain carnivora—kittens, pups, etc. Subsequent experiments have tended to show that herbivora are probably not susceptible to the poison, or, to say the least, are relatively insusceptible.

I next proceeded on a different track. It is difficult to understand why I did not try the following method first, as it would seem to be the one dictated by commonsense.

The end in view was to establish whether any connection existed between vomiting sickness and ackee poisoning, as appeared likely from the investigations into the Montego Bay outbreak.

In order to simulate as closely as possible the conditions under which, by this hypothesis, cases of vomiting sickness occur, I obtained some ackees which appeared good except for the fact that they were 'unopened' or had been forced open after being gathered.

These 'suspected' ackees were then boiled with water in the same way as that in which the fruit was used in the Montego Bay and outlying districts of Salt Spring, Granville, etc., in making the 'soup' or 'pot-water.' This ackee extract, as it may be called, was then filtered and used for experimental administration to animals. The extract thus obtained is a liquid of the colour of weak tea or thick soup with a layer of oily matter like melted butter floating on the surface. The animals employed, namely, three kittens and one dog, were fed with the extract, thus carrying out the conditions under which the pot-water is taken in the country districts.

The details of each of the four will be given:—

Kitten I. To all appearances perfectly healthy, five weeks old, weighing 500 grammes.

On March 28th, 1915, at 11 a.m., a dose of 5 c.c. of the watery extract was adminstered intragastrically. At 11.40 a.m. it suddenly vomited yellowish-brown watery matter, and seemed to be heavy and dull for half an hour or so, but was not comatose. During the afternoon it recovered, apparently completely, and by 4 p.m. was quite lively. The only other food given was fresh milk.

The next day, March 29th, it seemed to be quite well and had taken the milk provided. At 12 noon 10 c.c. of the same extract was administered in the same way. At 1 p.m. vomiting set in, and this was repeated during the next half-hour, after which the kitten

again improved and began to play about. At 3 p.m. another 10 c.c. was given. Vomiting again ensued, this time 45 minutes after administration, the animal became dull and somnolent, and could only be roused with difficulty; it could not walk but lay limply. Between 4 and 5 p.m. this somnolence deepened to coma, and death took place the same evening. No convulsions were observed. The total amount given was about equivalent to the extract obtained from one ackee.

At the post-mortem on the 30th the *liver* was noticed to be paler than normal, and the lighter parts were in patches; the *kidneys* were congested; the *bladder* was about half full of clear urine. The *stomach* showed a patch of slight congestion, no extravasation or petechiae. The *lungs* showed an infarct in the left, occuping about one-third to one-fourth of the lower lobe. No enlarged lymphatic glands were noticed. The vessels of the *meninges* were congested.

Histological examination yielded the following appearances:—

Liver: General engorgement, capillary and venous, hepatic and portal; in parts distinct extravasations among liver cells. Some oedema of stroma. Liver cells granular with vacuoles, sometimes four or five small, rarely very large; generally distributed, not more at periphery than towards the centre of the lobules. Nuclei generally stain well, but some degree of necrobiosis in parts showing the largest or most numerous vacuoles. Small microcellular infiltrations here and there.

With Flemming: Vacuoles (fat) in some parts much larger than in others, and in these the nuclei stain badly, in some cases being barely visible, and others showing karyolysis. Some cells, again, contain two or three vacuoles of considerable size, but nucleus is well defined; others contain several, in fact, the protoplasm is largely vacuolated, and the nuclei are fragmented and stain poorly.

Kidney: Hyperaemia, cortical venules distended. Some Malpighian tufts have dropped out, and, in their place, are red blood corpuscles; others show a lining of blood just within Bowman's capsule, and the Malpighian body has shrunk from the capsule. In nearly all there is a lining of fibrin or corpuscular débris between the glomerulus and the membrane. In the convoluted tubules the epithelial cell limits are badly defined, but the nuclei mostly stain well; the protoplasm is granular and the tubules often contain granular débris, which may be corpuscular detritus. In parts there is considerable necrobiosis, epithelial cell limits not definable, and nuclei badly stained. Some of the cells also show small fat granules at their bases, while occasionally the epithelium is distinctly vacuolated. In the straight tubules many of the nuclei stain poorly; the ascending limbs of Henle's loops contain débris similar to that mentioned above. There is a condition of general congestion, and in one or two spots small extravasations. No increase of stroma.

Pancreas: Vessels in interlobular tissue engorged. The epithelium shows some degree of necrobiosis, but not very marked; the islets of Langerhans show up well. Epithelial degeneration varies in different parts of the section; in some the cells appear almost normal, but in others they are granular, cloudy, and the

nuclei stain badly. In sections from another part of the gland the cells are vacuolated, and the stroma appears oedematous. No haemorrhages are seen.

Stomach : Nothing abnormal seen microscopically.

Spleen : Malpighian bodies prominent. Vessels are congested. There is oedema of stroma, possibly some increase, with small-celled infiltration in parts. In patches are areas of cells with poorly staining nuclei, and in these parts is also seen some débris, strongly suggestive of small extravasations, though definite corpuscles cannot be made out.

Lung : Intense congestion in parts, wedge-shaped as infarcts, and here the alveoli are largely occupied by fibrin, but some contain corpuscles. Alveolar walls here and there are broken down, so that spaces equivalent to four or five alveoli are filled with fibrinous débris, intermixed with which are occasional leucocytes and epithelial cells.

Giemsa stained sections show the presence, here and there, in the congested patch of organisms, diplococci and others (see later, after description of Kitten III). Flemming sections show no fat.

Heart muscle : Fragmentation of the fibres, and, in parts, some of the nuclei stain badly, while the fibres show coarse longitudinal striation with obscured transverse. Most of the fibres and nuclei stain normally. In two or three situations there are minute extravasations of blood between rows of fibres.

Thymus : Congestion of vessels, some oedema of stroma and minute extravasations are seen in a few places. Hassall's corpuscles well shown.

Cerebrum : Vessels of meninges congested ; also small vessels of the interior. Nothing else abnormal noticed. Nuclei generally stain well, and are normally situated in the cell. This tissue was not taken in Flemming's solution.

It will thus be seen that the characteristic symptoms of the so-called vomiting sickness appeared about one hour or so after the administration of filtered watery extract of ackee. After a small initial dose there was vomiting and rapid recovery; after a larger dose more severe vomiting, and after a third a repetition of the vomiting with drowsiness progressing to coma and death.

The post-mortem findings were typical of those seen in cases of human vomiting sickness, except that some micro-organisms (pneumococci) were found in the infarcted part of the lung.

These have not been mentioned in human cases in which infarction occurred, perhaps they were not present, possibly they were overlooked. I believe they were accidental in this case (see p. 49).

Kitten II. Quite healthy, six weeks old, weight 555 grammes. On April 20th, at 11 a.m., 5 c.c. of freshly prepared watery extract of unopened ackees was administered intragastrically. Within the next hour repeated vomiting occurred and the kitten was inclined to lie down. By 1 p.m. it was again lively and running about, and to all appearances had quite recovered. Some fresh milk was given.

At 2 p.m. another 7 c.c. of the extract was administered, and then followed the same symptom of vomiting, but more severe than after the first dose. The animal was drowsy, almost somnolent, but could be roused. Some milk was left for food during the night, and by the following morning the kitten appeared well again and was playful.

At 11 a.m. on the 21st, 10 c.c. of the same extract was administered; vomiting set in at 11.50 and the attacks were repeated; with intervals of improvement in the general state. At 2 p.m. another dose, the same amount as the last (10 c.c.) was given, and at 3 p.m. the vomiting was again severe, and the animal was very drowsy, the head nodding with sleep, the kitten frequently rolling over on to its side as with muscular relaxation in sleep, and then temporarily rousing up. 15 c.c. was administered at 4.10 p.m. and the animal died the same evening. The equivalent of about two ackees was given in all.

Autopsy at 10 a.m., April 22nd. *Liver* pale, possibly enlarged. The *kidneys* were congested, the *bladder* full of clear urine which on examination was found to contain albumen in small quantity, and after centrifugalisation a few epithelial casts and some red blood corpuscles were seen. The *Right Lung* contained an infarct occupying about one-third of the upper lobe; the *stomach* showed a little congestion and a few petechiae. The vessels of the *meninges* and the cortex cerebri were congested.

Histological examination of the various organs revealed the following conditions:—

Liver: Veins and capillaries congested, hepatic as much as, if not more than the portal. Capillaries between the rows of cells are engorged in parts, and extravasations may occasionally be seen between the cells. The cells are vacuolated and stain badly; vacuoles larger and more distinct towards the periphery than towards the intralobular vein, where the cells are more granular and vacuoles, if present, are smaller. Flemming sections show well-marked fat droplets, but the fluid has, unfortunately, not penetrated through the tissue, and the changes, therefore, are more marked at the periphery of the section.

Kidney: Vessels are congested, and adjacent to the glomeruli may be seen collections of red blood corpuscles. The glomeruli appear to be swollen, but, at the same time, do not entirely fill the spaces, and the intervals are occupied by blood corpuscles and fibrinous débris, while here and there glomeruli appear to have dropped out, and their places are occupied by blood. The nuclei of the glomeruli on the whole stain well. The convoluted tubules show granularity of epithelium; the cell limits are ill-defined, and some of them show small vacuoles at the base, but the majority of the nuclei have taken the stain well. The canals are often filled with granular material.

Flemming sections show considerable fatty degeneration of the epithelium of the convoluted tubules and the ascending tubes of the loops of Henle.

The stroma is more prominent in places, owing to the poor staining of the adjacent tubular epithelium. In many situations the stroma appears to be increased, but is not oedematous, and the nuclei stain well. In some of these parts a sort of mosaic pattern is produced owing to disappearance of the tubular epithelium, the place of the latter being taken by blood, and, where the epithelium remains, the tubules give the impression of being contracted by pressure of the stroma, and such tubules may be seen to contain corpuscles in their lumens.

Pancreas: Epithelium granular, but nuclei stain well; islets of Langerhans prominent. Some of the cells contain fat droplets (Flemming) especially at the periphery of the section. From the appearance of the cells those in the interior of the section would seem to contain fat also, but, unfortunately, the solution has failed to penetrate deeply. The vessels are engorged, the stroma is oedematous, and some degree of necrobiosis is present. No haemorrhages are seen.

Spleen: Stroma appears to be somewhat in excess of the normal; possibly it is oedematous, but the nuclei stain well. There are fairly extensive extravasations scattered through the section. Where the fibrinous and blood débris are greatest the cells appear necrotic and the nuclei stain poorly.

Stomach: Mucous membrane congested, and petechiae present near the cardia. Microscopically, there is an effusion of blood on the surface and amongst the gastric glands at one part of the section.

Lung: Vessels in parts congested; alveoli, on the whole normal, but, in places, some contain granular débris and shed epithelium. At one situation is a well-marked infarct, typically wedge-shaped and fading rather abruptly through a moderately congested zone to healthy lung tissue. No organisms of any kind can be seen; sections were stained for this purpose by Giemsa, by the Eosin-Gram-Weigert, and by the Picro-carmine-Gram methods.

Heart-muscle: Vessels generally congested, some engorged; the fibres are fragmented, minute extravasations are seen here and there. The nuclei generally stain well, and the striation of fibres is well preserved. Nothing abnormal otherwise; no granularity or vacuolization of fibres. No fat droplets seen in sections treated by Flemming.

Thymus: Vessels full of blood; small extravasations scattered through the gland tissue. Hassall's corpuscles well seen. The nuclei on the whole stain well, but at the sites of the extravasations and in certain lobules are areas of cells staining badly.

Cerebrum: Vessels congested, and in places there is a slight microcellular infiltration of the pia, but these are not very marked nor are they numerous. The nerve cells stain well and the nuclei are distinct almost throughout, but karyolysis in individual cells is not infrequent. This tissue was, unfortunately, not taken in Flemming.

Cerebellum: Shows the same state of things; except for localized small masses of microcellular infiltration of the pia and some oedema, with congestion of the vessels, nothing abnormal is detected.

Kitten III. Six and a half weeks old, quite healthy, weighing 585 grammes.

At 12 noon on April 23rd, 8 c.c. of fresh extract, prepared as before, was administered. At 12.45 p.m. the kitten started to vomit, and did so three times between this and 3 p.m. It seemed inclined

to lie down before the attacks of vomiting, and each time recovered partially and walked about the room. By 4 p.m. it appeared quite well and was playful. The following day, April 24th, there were no signs of disease, in fact the animal was fairly lively. At 10.45 a.m. 20 c.c. extract was given. For 45 to 50 minutes there was no apparent effect; it then became restless and continually lay down; vomiting occurred four times between 11.30 a.m. and 1 p.m. Drowsiness supervened and the animal could only be roused with difficulty. On attempting to walk it was very unsteady. At 1 p.m. another similar dose was given; vomiting ensued almost immediately (very little of this last dose can have been retained) and in a quarter of an hour the animal became comatose, and died at 1.35 p.m. There were no convulsions. The extract from two ackees was given altogether.

Autopsy at 1.50 p.m. The *lungs* showed minute patches of congestion but no definite infarcts. The *stomach* was contracted and contained some of the extract with small lumps (curdled milk; the food throughout had consisted of fresh milk only). The *liver* was macroscopically normal, possibly a little paler in patches. *Kidneys* showed surface veins congested, and there was also some congestion on section. The vessels of the *brain* were full, as in the previous cases.

Histologically:—

Liver: Vessels engorged, portal as well as, but possibly a trifle less than the intralobular; extravasations in several situations, especially towards the centre of the lobules, and there were capillary haemorrhages between the rows of liver cells. These cells showed marked vacuolization, and this condition was more noticeable towards the portal region of the lobules. Some sections showed extensive haemorrhage and destruction of liver tissue. Cell-nuclei as a rule stained well, but many showed some degree of karyolysis.

Flemming fixed sections showed a wide-spread fatty change, often as many as twenty or more black droplets in a cell, often only five or six, but larger. The fat is fairly evenly distributed, but in some lobules the periphery is perhaps a little more affected.

Kidney: Capsule not thickened; subcapsular and cortical vessels very congested and distended with blood. Small extravasations in cortex and parts of medulla. Glomeruli are swollen, but in most instances there is a space between the glomerular tuft and Bowman's capsule, and in some cases this space contains fibrinous and corpuscular débris. The convoluted tubules show swelling of the epithelium, with granularity, and the lumens are, many of them, occupied by granular material. Several of them contain epithelial and granular casts, and there are small vacuoles at the bases of several of the cells. The stroma, as in No. II, is in parts of the medulla very prominent, where the epithelium of the

tubules cut transversely seems to have disappeared, and the canal is either empty or contains red blood corpuscles and débris. The nuclei of the stroma cells stain well, but those of the epithelium vary. In parts, there is a small-celled infiltration of the stroma, but this is infrequent. Many of the tubules on cross section show only one or two cells remaining, and the canal contains shed cells, fibrinous débris, and sometimes red blood corpuscles, revealing transition stages between those with swollen epithelium and granular contents, and those from which the epithelium has disappeared and whose canals are either empty or contain red corpuscles and granular or fibrinous débris.

Flemming sections show that the cells of many of the convoluted tubules and of the ascending loops of Henle contain fat, often in large drops occupying almost the entire cell; in others there are several small droplets, and the nuclei are still visible. The fat in No III is rather less than in No. II, but death was more acute, and very little excretion can have had time to take place.

Pancreas: Vessels congested, stroma somewhat oedematous, necrobiosis slight. Sections stained by haematoxylin and Hansen's modification of van Gieson do not show well-marked nuclei, though the islets of Langerhans are prominent. Giemsa-stained sections do not show sufficient differentiation. Those stained by Flemming and safranin show distinct nuclei, and occasional cells have fat-stained contents. In one part there is a fairly extensive haemorrhage, apparently interstitial in origin, but causing considerable destruction of cells.

Spleen: General congestion of vessels and a few small areas of extravasation are seen; oedema of stroma, which appears to be rather in excess of what is usually present, possibly owing to the swelling and oedema. In the extravasated areas the cells show some necrosis, the nuclei stain poorly, and exhibit some karyolysis; in other parts the cells take the stain well.

Stomach: Macroscopically, congestion, but not marked, though extensive in superficial area. Microscopically, nothing of importance noted.

Lung: Vessels congested; alveoli mostly normal, but here and there are some containing red blood corpuscles, and in other places some fibrinous and granular débris, with shed alveolar epithelium. In the neighbourhood of such are distended alveoli (compensatory emphysema). The bronchioles in parts also show granular matter, with mucus and entangled epithelium and a few leucocytes. No pneumococci or other organisms seen.

Heart-muscle: Minute capillary haemorrhages, here and there, between the muscle fibres. Striation fairly well preserved and nuclei stain well; very little fragmentation.

Flemming-stained sections show black fat granules in some of the fibres; the degeneration, however, does not appear to be extensive, but, unfortunately the solution has not penetrated well.

Thymus: Apparently normal.

Cerebrum: Congestion of vessels of pia, and engorgement of the cerebral capillaries generally, which, here and there, appear to have given way. The pia is oedematous.

Flemming sections reveal a peculiar condition: several of the capillaries show minute droplets stained black, and some of the nerve-cells show a similar condition of the protoplasm. In this animal the capillaries are more affected than the cells (see No. IV).

Cerebellum: Congestion of vessels as in the cerebrum, and in addition, a few of the cells in the sections treated by Flemming's method show black droplets; some of the capillaries also contain these droplets. Nothing else abnormal seen.

In this case we see the symptoms typical of vomiting sickness following rapidly after administration of 'pot-water,' and the post-mortem findings, macroscopical and microscopical, are identical with those of a human case.

In the case of Kitten I, one might, in fact one would, be inclined to infer that death was due to pneumonia, but though, of course, this aspect must be considered, I am not inclined to adopt this view for the following reasons:—

1. The organisms were not very numerous.

2. They were confined to the infected area, which was a small one.

3. The animal had no cough and no apparent respiratory embarrassment before death.

4. The animal died rapidly, with typical vomiting sickness symptoms. It had been to all appearances quite well and running about at noon on March 29th, and died the same evening (about six hours after).

5. There was no enlargement or affection of the bronchial glands.

6. The other organs, liver, kidneys, etc., showed the typical pathological appearances found in cases of death from vomiting sickness.

7. Nos. II and III showed the same changes in the other organs as No. I, but had no pneumococci in spite of considerable congestion of the pulmonary vessels and some of the alveoli, and a definite infarct in the case of No. II.

8. The organisms were not found in the blood-vessels of any other organ, that is, a condition of septicaemia was not present as one would reasonably expect in death arising so acutely, if due to this cause (pneumococcal infection).

The ackee season being practically over, it was becoming a difficult matter to obtain the fruit. However, as we had remaining from the last experiment the extract equivalent to four ackees, we thought it advisable to administer this to another animal, a pup, and if this amount was, as we expected, insufficient to cause death, it would be advantageous to destroy it at an interval of some hours after the last dose and see what may be called an intermediate stage of the disease.

The results were most instructive.

Dog. Female, aged two months; healthy and well nourished; weighing 1850 grammes.

On the 10th May, at 11.40 a.m., 25 c.c. of the extract was administered intragastrically. No symptoms showed themselves till 1 p.m., when the animal was noticed to be unnaturally sleepy; no vomiting had occurred. It had had a good feed of milk a short time previously, and consequently absorption was probably slower. By 2 p.m. the drowsiness was passing off and the animal was obviously recovering. Another 10 c.c. was given. Within two hours the drowsiness had returned, but this was not deep and the puppy could be roused; in fact, it took some food in which about 15 c.c. of the extract was mixed.

The next morning, though not very lively, the animal appeared well. At 11 a.m. 15 c.c. was adminstered; at 12.20 p.m. it vomited a considerable amount of yellowish watery material, and seemed to be drowsy. No other symptoms occurred between this and 2 p.m., when 25 c.c. was given. After a similar interval ($1\frac{1}{4}$ hours) vomiting again occurred, the animal was somnolent, lying down all the time, drowsy but not comatose. At 4 p.m. the remainder (about 20 c.c.) of the extract was given. In all, as stated, the puppy took the equivalent of three to four ordinary ackees. During the evening it vomited, and, though it took some of the milk provided for it during the night, the animal did not appear well the following morning and was disinclined to move.

I think it was more than probable that it would have died, as it was now refusing all food, but as we had no more extract left, and, as already mentioned, we wished to observe an intermediate stage of the affection, the puppy was rapidly chloroformed at 11 a.m., and the autopsy begun before five minutes past.

Macroscopic appearances post-mortem:—

Liver very pale, almost boxwood in appearance throughout; the *stomach* showed a small congested area towards the cardia, no ulceration; the upper part of the *duodenum* was very slightly congested; the remainder of the intestine was normal. The *mesenteric glands* were enlarged and some of them markedly hyperaemic. *Kidneys* showed a dark surface and the cortex was congested, the pyramids were pale. *Spleen* and *pancreas* showed no obvious change. *Lungs* were generally congested, a few

petechial patches, no infarcts seen. *Heart-muscle* apparently normal. *Brain*, both cerebrum and cerebellum, showed congested vessels on the surface, and possibly also on section.

The *bladder* was full of pale yellow clear urine. No definite albumen reaction was given, but the centrifugalised deposit showed a few hyaline casts and several epithelial and granular ones.

Histological examination:—

Liver: Congestion of vessels, veins and capillaries, both hepatic and portal. The capillaries between the columns of liver cells are full, and, in many cases, they appear to have given way, and there are collections of red blood corpuscles and granular and fibrinous débris between the cell rows. The nuclei throughout have taken the stain moderately well, but nearly all the cells show vacuolation in all stages, from the very minute to a condition in which practically the whole cell is vacuolated. There are in one or two patches small collections of micro-cellular infiltration. Flemming sections show widespread fatty degeneration, the cells containing very many small droplets of fat universally distributed over the lobules, wherever the fluid has penetrated. In a few situations these small droplets have coalesced to form larger masses, but in the majority of cells they are minute, and so numerous that not infrequently the nuclei are obliterated.

Kidney: Vessels congested, and in some situations the engorgement is such that small extravasations have occurred. The glomeruli (as in kitten III) are separated from the capsule by an interval which, in many cases, contain red blood corpuscles, or fibrinous residue. In some of the cells of the glomeruli small vacuoles are seen, but the nuclei on the whole stain well. Some of the tufts appear to have ruptured. The epithelium of the convoluted tubules is swollen and granular, and the canals contain epithelial and granular casts, and occasionally blood. The limits of the cells are often obscured, but on the whole the nuclei are fairly clear; in some cases karyolysis is present. Flemming sections show fatty degeneration of the cells of the convoluted tubules and ascending limbs of the loops of Henle, less of the descending. The fat is situated at the base of the cells. The condition is widespread, but in minute droplets, rarely coalesced to larger ones. In a few instances droplets can also be seen in the glomeruli and cells of Bowman's membrane.

Pancreas: Vessels moderately full of blood; some of the interlobular and interacinar capillaries congested; no extravasations seen. Stroma oedematous; practically no necrobiosis; in one or two very localised situations the cells are indistinct and the nuclei have not stained well, but otherwise, in sections stained by Hansen's method or Giemsa, the gland appears normal. Flemming sections show very widespread, but not severe, fatty degeneration. By this is meant that very many cells (perhaps the majority) show minute fat droplets, but in very few have they become confluent. The cells of the islets of Langerhans are not exempt, but they are less affected than the gland cells proper.

Spleen: Vessels engorged; stroma appears to be in excess of the normal. Nuclei stain well. In various situations extravasated red blood corpuscles are seen, or fibrinous and granular blood residue. Flemming sections show that many of the cells contain minute fat droplets; they are distributed widely throughout the section, but nowhere are they large. This condition has not been noticed in any of the other animals.

Stomach: Nothing much abnormal seen. Small extravasations (petechiae)

at the mucous surface, here and there, and localised hyperaemia. Patchy infiltration of the submucosa, with congestion of capillaries of this layer and the base of the mucosa.

Lung: Small patches of congestion and bronchopneumonia seen; some alveoli contain red blood corpuscles, shed epithelium and a few leucocytes, the neighbouring alveoli being enlarged and distended to compensate. The rest of the organ appears normal, except for capillary congestion. No infarcts present and no micro-organisms seen.

Heart-muscle: No fragmentation; transverse striation well preserved; nuclei stain well. Minute extravasations in parts between the muscle fibres. In the deeper layers of the visceral pericardium, in fact, almost subpericardial, there is a more extensive haemorrhage, which has led to some destruction of the subjacent muscle fibres. This may possibly have been caused when the piece of tissue was taken.

Thymus: Except for some microcellular infiltration of the stroma in a few situations, nothing abnormal seen in the sections. Hassall's corpuscles are not numerous, but, where present, they show up well. The capillaries in parts are congested, but no extravasations are seen.

Mesenteric lymphatic gland: Congestion of vessels and general diffuse hyperaemia. Extravasations of blood scattered in various parts of the section. These are in most instances, but not in all, seen in the interior of the lobules of the gland tissue. The nuclei of the gland cells, except in the area of extravasation stain well. There is some oedema of the stroma.

Cerebrum: The pia appears oedematous, and the vessels are congested. The capillaries of the interior of the section are also full, but no haemorrhages are visible. Flemming sections reveal the curious fact that many of the nerve cells contain minute droplets stained black by the osmic acid. Many of the capillaries also contain fat; in fact, the picture suggests a mixture of degeneration of nerve cells together with (possibly arising from) an embolic condition of the capillaries. I am not at all convinced, however, that these two processes are not distinct, for in parts the capillaries show these droplets while the cells affected may be some distance away, those nearest to the capillary apparently not being affected; on the other hand, in some situations, several cells may show these changes while the adjacent capillaries do not contain the droplets.

Cerebellum: Similar to the cerebrum in congestion of vessels of the meninges and interior, with oedema of the pia. No haemorrhages seen. It may be noted that the cells of Purkinje in some instances show fragmentation of the nucleus, while in a few, the protoplasm is very granular and no nucleus can be made out at all. The majority, however, stain normally. Flemming sections show capillaries containing fat droplets as in the case of the cerebrum, but very few of the cells appear to be affected. Those that are affected are mainly in the close neighbourhood of the capillary from which some emigration of corpuscles had occurred, or whose wall has given way.

It is hoped that further experimental work may be undertaken during the coming winter. This will come within the domain of the chemist rather than that of myself. The aim will be to separate from an aqueous extract of the ackee the toxic principle or principles, and to test the effects of the various isolated constituents on animal subjects.

I am greatly indebted to Mr. Simmons for agreeing to undertake the first part of this work.

VII. ACKEE POISONING

Report of a recent Case of Poisoning by the Fruit of the Ackee (*Blighia sapida*).

While writing up the last section a most interesting occurrence took place in a country district thirteen miles from Kingston, which not only goes far towards supporting my contention but practically affords the solution of this difficult problem.

On Thursday evening, August 19th, 1915, a family of eight, all of whom were at the time in good health, partook of a meal of ackees taken from a tree which had been 'blown by the hurricane' of the previous week. About two hours later almost every one of the eight complained of feeling sick. Some home remedy (nature not known) was given to all, and they experienced some relief. The following morning they still felt ill. During the day three of them began to vomit, and one, aged 42 years, suffered from convulsions, became unconscious and died the same evening. The others were seen by a medical man, Dr. S. C. Ormsby, who treated them for 'irritant poisoning' and reported the cases as suffering from ackee poisoning. They made good progress.

One member of the family denied that any ackees had been eaten subsequent to the meal on the 19th, but others stated that they had some more on the following day. This would explain the nature of the symptoms in the three individuals mentioned.

The one who died had drunk some of the 'soup.'

During the succeeding six days some of the family still ate ackees for their meals on and off. On Wednesday, 25th, at 6 p.m., M. S., female, 22 years of age, drank the soup and had some of the meal of boiled ackees. About 8 p.m. she vomited, and slightly improved. Later (about 10 p.m.) she had a return of the vomiting, became convulsed, lost consciousness and died shortly after midnight. Another member of the family was also taken ill, but recovered after vomiting.

Having received a telegram relative to this case, I went to the place and carried out a post-mortem examination in the presence of Dr. Ormsby, at noon on the 26th.

The body was that of a well-nourished woman whose age was stated to be 22 years. There was no jaundice or discoloration of conjunctivae; there was no rash, no lice or ticks.

Prior to starting on the dissection, I made a lumbar puncture and obtained a few drops of cerebro-spinal fluid. Two tubes of nasgar were inoculated, and smears were made. The fluid was not present in excess, nor was it under pressure.

Macroscopically :—

Brain : The meninges were congested, and there was some serous effusion over the arachnoid, at the convexity and at the base. Fluid was present in the ventricles, but not in excess. There were no macroscopic signs of disease on section of the brain tissue, except a few petechial points.

Lungs : Congested ; no true infarcts, but one part in the left lower lobe appeared to be a little more deeply congested than the surrounding parts.

The lung tissue was frothy, crepitant, and floated in water. The trachea contained a little frothy mucus. Larynx normal. Bronchial glands not enlarged.

The *Myocardium* was possibly a little pale, but the heart appeared otherwise normal ; there was no valvular defect. A few small petechiae were seen on the visceral pericardium at the base of the right ventricle.

Thyroid normal in aspect.

Stomach : Showed slight congestion, especially towards the pylorus, where two or three petechiae were visible.

The contents were very small in quantity, and consisted of frothy, grumous material.

The *Duodenum* showed congestion also, but even less marked than that of the stomach.

Nothing abnormal found in the intestines, no worms present.

The *Liver* was of a yellowish red colour, with pale patches, and looked 'fatty.' The *Spleen* was small but apparently normal, except that the fibrous connective tissue seemed to be in excess. The *Pancreas* was distinctly congested, but there were no visible haemorrhages, and the consistence seemed normal. The *Kidneys :* stellate veins very prominent, capsule transparent, smooth, not adherent. On section of the kidney tissue, pale streaks were seen in the cortex, and the bases of the pyramids were congested. The *Mesenteric glands* were enlarged and some of them hyperaemic.

Microscopically :—

The spinal fluid smears showed no organisms and very few cells, and these were nearly all mononuclear. The culture tubes revealed two colonies of *Staphylococcus albus* in one of them, the other remained sterile. This growth was probably a skin contamination in making the puncture, as the autopsy was performed in the bush, and not under the best bacteriological conditions.

Cultures of the heart-blood remained sterile, and no parasites were seen in the blood smears.

Small pieces of the various organs and tissues were taken (*a*) in alcohol, (*b*) in Flemming's solution, and embedded in paraffin. The following pathological changes were seen on examination of the sections :—

Cerebral Cortex : The pia is oedematous and the vessels congested. There is congestion of the vessels of the brain substance, but not in a marked degree. The nerve cells, as a rule, have taken the stain well, and their nuclei are normally situated ; occasionally there is displacement of nucleus, with poor staining, and some karyolysis.

Flemming stained sections show that several of the capillaries and larger

vessels contain fat droplets, but these more often appear to be in the cells of the vessel wall than actually within the lumen as emboli.

Many of the nerve cells are seen to contain black droplets (fat). These may be so numerous as to nearly fill the cell, but in the majority the nucleus is still visible; when the number of droplets is large the nucleus may be displaced.

Cerebellum: This shows similar congestion of vessels, but less marked than in the cerebrum. The cells have taken the stain well almost everywhere, but in a few situations the cells of Purkinje show the nucleus eccentric and fragmented; while in one or two no nucleus can be made out at all, and the cells appear necrotic.

Flemming-fixed sections show the same changes as have been described in the cerebrum, but not so extensive nor so marked.

Lung: Marked congestion, vessels all engorged. In parts the alveoli are distended and contain red blood corpuscles, leucocytes, and shed epithelium. The alveolar walls are broken down in places, so that large irregular spaces are produced containing the same elements. The mucous membrane of the bronchioles is swollen and the epithelium is shed into the lumen.

In Giemsa stained sections a few cocci are visible here and there, a post-mortem or accidental contamination.

The above conditions are seen in the congested parts of the lungs; sections from other parts show little except filled capillaries and some shedding of alveolar epithelium.

Flemming treated sections show fat droplets of varied size in various scattered situations. They are mainly present in the walls of the alveoli (pulmonary capillaries), and only rarely have they coalesced to form large drops.

Heart-muscle: The epicardium appears normal; the subepicardial vessels are engorged. Occasionally small microcellular infiltrations of the intermuscular connective tissues are seen, and a similar infiltration beneath the epicardium. The muscle fibres do not show any abnormality, except a few in which minute vacuoles are visible. The transverse striation is well preserved, and there is little or no fragmentation of the fibres. The nuclei stain well on the whole, but here and there are some which show karyolysis and have stained badly.

Flemming sections show that several of the fibres contain fat droplets; some only a few, others are nearly filled with them.

Liver: No alteration seen in the capsule. The vessels in parts exhibit a certain degree of congestion, but no haemorrhages are seen. The periportal connective tissue shows some apparent increase, but this may be due to contrast with the vacuolated liver cells.

The liver tissue itself is almost unrecognizable owing to the intense and widespread fatty metamorphosis. Some of the cells of the parenchyma (the minority) show a number of small vacuoles, but nearly all are distended by a large fat drop, the cell nucleus being thrust to the periphery. The nuclei of some of the cells take the stain well, others are barely visible, while others, again, show karyolysis. The fatty change is intense, and distributed over the whole section almost equally; in some cases, however, the fat drops in the neighbourhood of the portal vessels are larger than those in the interior of the lobule, but this is by no means always the case. Few recognizable liver cells are left. This intense change may be explained by the fact that the patient had been taking ackees, on and off, during the week, and had been ill after the meal a week previously, and had taken the 'soup' (extracted poison?) again the same evening before she died.

Flemming sections do not reveal anything further, except to prove that the vacuolization is due to fatty metamorphosis.

Kidney : General congestion, and in one part of the section actual extravasation has occurred. The glomeruli are swollen, and in a few, between the tuft and Bowman's capsule, there is granular (corpuscular ?) débris. The cells of many of the glomeruli are necrobiotic, and into the glomerular spaces, in some instances, the lining cells of Bowman's membrane are shed. The intertubular connective tissue is slightly increased. There is an intense parenchymatous nephritis ; many of the convoluted tubules show disorganisation of structure—the cell-limits are undefined, the nuclei are hardly recognisable (in parts they are not visible at all), the cells are granular, vacuolated, and necrobiotic. Many of the tubules contain red blood corpuscles or their residue, and in some the secreting epithelium has quite disappeared. In others, the lumen is occupied by blood, or by granular degenerated epithelium. The lining epithelium of many of the convoluted tubules and of the ascending limbs of the loops of Henle show extensive vacuolization, especially marked at the bases of the cells. The descending limbs of Henle's loops have not escaped, but they are less affected.

Flemming sections show extensive fatty metamorphosis. This has barely affected the Malpighian tufts at all, a few black dots only are seen in them, and in the cells of Bowman's capsule occasionally. But in the epithelium of the convoluted tubules and the ascending limbs of the loops of Henle, the change is very marked ; less, but still extensive, in the descending limbs.

Spleen : The capsule is increased somewhat in thickness, and the interstitial tissue throughout the section is in excess of that normally present. There is general congestion, but no haemorrhages are seen ; here and there, however, are patches of what appear to be fibrinous and possibly corpuscular débris scattered amongst the parenchyma cells.

Flemming sections reveal minute droplets of fat scattered in various parts of the section. They are present in the cells of the parenchyma, but to a considerably less extent than in the fibrous trabeculae, though nowhere are they numerous.

Pancreas : Vessels congested, but no extensive haemorrhages seen. There are one or two spots where small extravasations have occurred. The interlobular connective tissue appears increased and possibly somewhat oedematous. There is a similar condition of the interacinar connective tissue.

The glandular epithelium shows very extensive necrobiosis. The cell-limits are badly defined, the nuclei in many cannot be seen, in others there is karyolysis. In fact, in a few situations only is the normal pancreatic epithelium visible. Many of the cells appear to contain minute vacuoles. The islets of Langerhans are also affected, and can rarely be distinguished in the tissue. Flemming treated sections reveal the fact that barely any normal pancreatic tissue is left, the fatty metamorphosis and necrobiosis are so marked. The nuclei are visible, but otherwise the cells appear to be filled by black-stained fat droplets, which often have coalesced so as to practically obliterate the cell.

Lymph-gland (mesenteric) *:* There is general congestion of the gland ; the vessels are engorged, and in one or two situations the capillaries appear to have given way. The stroma is oedematous. The majority of the cells stain well, but in many parts are areas where the cells are necrosed—they are granular and have barely stained at all. Such occur in considerable patches, but also, in other parts, are intermixed with cells which have stained normally.

In this case, then, we have a definite history of a patient in good health partaking of a meal of ackees from a bruised limb. She, with other members of the family, suffered from vomiting, and

recovered. A week later another meal was prepared with fruit from the same tree. The patient drank the 'soup' and also ate some of the solid. Two hours later the symptoms made their appearance and ran their course to a fatal termination in six hours or so, and at the post-mortem the changes which have been described above and in the last section were revealed. In this case the term 'Vomiting Sickness' was not used from first to last, but the case showed typically the onset, course, and pathological changes of that disease.

Before finally summing up the points treated in detail in this paper, a brief consideration of certain peculiarities and characteristics of the affection may be given. This will demonstrate the similarity—a similarity which, in my opinion, amounts to identity—between vomiting sickness on the one hand and the experimental results in animals of administration of ackee extract and the clinical case of definite ackee poisoning on the other:—

(1) Peculiar seasonal prevalence.

(2) Its confinement to Jamaica so far as is known.

(3) Sudden onset of symptoms in apparent good health, and in the well-nourished as in the emaciated.

(4) The rapid and complete recovery of non-fatal cases.

(5) Affection of several members in one house or close neighbours in a settlement.

(6) Its vastly greater preponderance in children.

(7) No preference as regards sex.

(8) White children practically never attacked, East Indians rarely.

(9) The pathological changes set up.

(1) *Peculiar Seasonal Prevalence.* The disease corresponds exactly with the main ackee season, when other fruits and natural foods are relatively scarce. This year the ackee season has continued longer than in previous years in my experience; instead of ending in March or early in April the fruit has been abundant till well on in May; and, owing to an exceptionally good season of rains at unusual times, some trees are even bearing now (August). Vomiting sickness cases have been reported this year in greater numbers than last, and over a more prolonged period. This correspondence between the ackee season and vomiting sickness cases is borne out by my records for three or four years past.

(2) *Limitation to Jamaica.* As Seidelin states (p. 455 of his report): 'The Government of Jamaica made enquiries in a circular 'letter to the authorities of other West Indian Islands, with regard 'to the possible occurrence of vomiting sickness in other parts of the 'West Indies. Only two of the answers are quoted by Ker in the 'Annual Report (1906), the others being in the negative. The one 'positive answer is from Finlay, who mentions an outbreak of 'twenty-five similar cases with five deaths amongst soldiers in 'Cuba; he suggests that the disease is cerebro-spinal meningitis. 'No particulars are given in the quotation. The other positive 'answer deals with a disease (in Haiti) which has obviously no 'essential resemblance to vomiting sickness.'

I have made enquiries from those who are in a position to know, and I am told that ackees do not grow, at all events to any extent, in any other island, and the limitation of the disease to Jamaica would thus find explanation. One or two trees only are growing in other islands, but they are looked upon as curiosities and are not used for food.

It is true that some ackees are shipped to Colon from Jamaica, but only such as are ripe and open or which open within twenty-four hours of leaving Jamaica.

(3) *Sudden onset of symptoms in apparent good health* and in well-nourished, not necessarily emaciated, subjects. The symptoms, as has been pointed out more than once in the foregoing pages, are those of an acute intoxication, and those symptoms would depend not so much on the general well-being of the subject as on the dose of the poison and the condition of the stomach (whether empty or full) and its readiness for absorption.

(4) *The rapid and complete recovery of non-fatal cases.* This is practically answered in the last paragraph; an acute vegetable poison is taken in small quantity, is got rid of by vomiting, and the patient completely recovers. This, one sees, for example, in mushroom poisoning.

(5) *Affection of several members practically simultaneously in one house*, or close neighbours in a settlement. These facts are both well supported in the Montego Bay report. Several members are affected in one house because they partake of a similar meal, or the same articles of diet are cooked up together and shared in common.

The graded acuteness of symptoms mentioned in cases where the adults ate the solid, the older children the semi-solid and absorbed watery extract, the younger ones the soup or pot-water, further bears this out.

Close neighbours in a settlement are affected because the trees are in and about the settlement and all share in the produce.

(6) The vastly *greater preponderance in children.* This has been shown in my previous reports, and spoken of in Section III. This is explained by the fact that they are given the pot-water, the most toxic part—in short, an extracted poison—and that the lethal dose of a poison is far smaller in a child, generally speaking, than in the case of an adult; and, lastly, adults know the risks of eating unopened ackees, while children naturally do not.

(7) *No preference as regards sex.* One would not expect a vegetable poison to exercise any sex-selective powers.

(8) *Attacking the West Indian native in much greater numbers than East Indian or White.* The coolie diet consists mainly of rice and split peas, often in the form of curry. They are also fond of green fruit, such as green mangoes, guavas, jack-fruit. They never eat pork or beef, but like goat when they can get it. After they have been in Jamaica for some time, some of them take gradually to eating salt-fish, but never to the same extent as the West Indian native.

They very rarely indeed eat ackee; a few may do so after they have served their time and are out of their indentures, and have settled in the island, but even then it is not a common article of diet with them.

One who has had a good deal to do with the East Indian in Jamaica tells me that though he believes, as just stated, that the older coolies occasionally use ackees, he himself has never yet seen them eat it.

The White buys his ackees in the market, where he can see and select his purchases; or, greater safeguard still, many of them will only eat ackees which are gathered carefully off their own trees.

(9) *The Pathological Changes set up.* These have been already dealt with, and need not be repeated. Some of the specimens from previous cases of vomiting sickness, those from the animals treated with ackee extract, and those from the case of ackee poisoning

described above, could not be distinguished from each other under the microscope, the changes set up are similar. Variations between cases are explicable by the poison acting more particularly on one organ. Thus, the congestion and even haemorrhages which occur may affect particularly the pancreas, for instance, and from this point of view two cases of vomiting sickness reported to me this year in which acute haemorrhagic pancreatitis was found might possibly have after all been cases of true vomiting sickness, the poison acting mainly on this organ. In others the liver may show marked changes due to absorption of the poison in larger doses from an empty stomach, and so on. There is no need to labour this point.

VIII. SUMMARY

1. The term 'Vomiting Sickness' has been used for many years as a comprehensive name for various diseases, including cerebro-spinal meningitis, gastritis, gastro-enteritis, worms, malaria; in fact, practically any disease occurring in the cooler months and associated with vomiting and convulsions.

2. During the last ten years opinions have been expressed to the effect that there is an affection called vomiting sickness whose course of symptoms and post-mortem changes are not those of any known disease.

3. The death rate from this affection is exceedingly high, 80 per cent. to 90 per cent., and a fatal termination occurs in a few hours.

4. The first systematic investigation into the affection was undertaken in 1912 by Captain (now Major) T. J. Potter, R.A.M.C., who came to the conclusion 'that the majority of deaths ascribed to the so-called vomiting sickness are due to yellow fever.'

5. To this succeeded the 'meningitis era,' a recrudescence of the older idea that some cases at all events included under the term 'vomiting sickness' died from cerebro-spinal meningitis.

6. Seidelin's investigation took place the following year (1913), but, though he was the first to give a detailed description of the morbid anatomy, he did not succeed in solving the question of causation. He showed, however, that there was a definite unexplained condition comprising the majority of cases reported as vomiting

sickness, and that the condition was neither yellow fever nor cerebro-spinal meningitis.

7. Investigations into a typical and severe outbreak at Montego Bay in February, 1915, revealed the fact that in a majority of the cases in which a reliable history was obtainable, ackees formed part of the last meal taken in health, and that this article of food could not be excluded in a single case.

8. Persons taking the 'soup' or 'pot-water' made with ackees in certain conditions showed the most acute symptoms; the onset occurred in two hours, and death nearly always resulted.

9. 'Salt-fish,' a frequent article of diet, is in the country parts used as a euphemism for 'salt and ackee.'

10. Ackees under certain conditions are undoubtedly poisonous; among such conditions are: (i) Unopened ackees; (ii) ackees picked from a decayed, bruised, or broken branch; (iii) ackees which have not opened naturally, but which have been forced open; (iv) ackees with a soft spot in an otherwise apparently sound fruit.

11. Much of the poison is extracted by boiling with water.

12. The symptoms of a case of typical vomiting sickness are: Initial vomiting (gastric in origin) coming on in apparently perfect health; a period of a few hours' improvement, succeeded by secondary vomiting (cerebral) rapidly followed by convulsions, coma, and death. The average total duration of illness is twelve and a half hours.

13. Initial, or secondary, vomiting, or convulsions may be absent, but not in a large percentage.

14. Recovery, so far as I am aware, has never occurred when once convulsions set in or coma; and, as a corollary to this, in no cases which recover are convulsions seen.

15. The affection is largely one of childhood, and shows no predilection for sex.

16. A reasonable interpretation of the symptoms is: Some poison is taken, or some substance which acts as a poison after it enters the stomach. If the initial vomiting is sufficient to get rid of this substance no further symptoms occur and recovery rapidly ensues. If this is not the case, there is an interval—a more or less quiescent period of absorption—after which there follow symptoms due to the action of the poison on the higher centres—secondary (cerebral) vomiting, convulsions, drowsiness, coma, and death.

17. In rare instances the cerebral symptoms are those first noticed (convulsions, drowsiness, coma); there is no preceding vomiting—the so-called 'vomiting sickness without vomiting.'

18. The pathological changes consist chiefly of general hyperaemia and a tendency to haemorrhages in various organs, fatty metamorphosis especially in liver and kidneys, and necrobiotic changes of epithelia in liver, kidneys, and pancreas.

19. Micro-organisms are rarely found in true vomiting sickness cases, and, when present, are probably accidental and have no pathological significance.

20. Intragastric administration of an extract, made by boiling unopened ackees with water, produced in three kittens and one pup the symptoms and pathological changes seen in cases of vomiting sickness.

21. A case of ackee poisoning in a human subject exhibited the same symptoms, course, and post-mortem changes, macroscopical and microscopical, as (*a*) human vomiting sickness cases, and (*b*) animals to whom an aqueous extract of unopened ackees had been administered.

22. The characteristics of 'vomiting sickness,' viz. :—

(i) Peculiar seasonal prevalence;
(ii) Its confinement to Jamaica, so far as is known;
(iii) The sudden onset of symptoms in apparent good health, and in the well-nourished as in the emaciated;
(iv) The rapid and complete recovery of non-fatal cases;
(v) The affection of several members in one house, or close neighbours in a settlement;
(vi) The vastly greater preponderance in children;
(vii) The absence of preference as regards sex;
(viii) The rarity of occurrence in White children and East Indians;
(ix) The pathological changes induced;

all find explanation in the view that the condition is an acute intoxication by unopened or unwholesome ackees—the fruit of *Blighia sapida.*

In conclusion, I desire to express my acknowledgments to the Hon. Dr. J. Errington Ker, Superintending Medical Officer, for allowing me to have access to the records of the department of which

he is the head; to the Hon. P. C. Cork, C.M.G., Member of the Legislative Council, late Colonial Secretary of Jamaica, for providing me repeatedly with ackees for experimental purposes; to Dr. H. W. Catto, M.B., B.S. Lond., my colleague, the Assistant Bacteriologist, who has so carefully carried out the irksome task of cutting the sections of the various tissues; to Dr. G. W. Thomson, District Medical Officer, Montego Bay, without whose help I could not have made so thorough an investigation into the outbreak there; to Mr. R. Simmons, F.I.C., Deputy Island Chemist, for the preparation of ackee extracts; and, lastly, to Mr. R. S. Martinez and Mr. Valdes, to whose skill are due the micro-photographs accompanying this paper.

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Table I.—Vomiting Sickness.

All cases reported of which the histories were reliable.

		Under 1 year	1-2 years	2-3 years	3-4 years	4-5 years	5-6 years	6-7 years	7-8 years	8-9 years	9-10 years	10-11 years	11-12 years	12-13 years	13-14 years	14-15 years	15-16 years	16-17 years	17-18 years	18-19 years	19-20 years	Over 20 years	Total
Fatal Cases	Male ...	2	5	15	11	10	10	12	6	7	1	2	3	4	1	—	—	—	1	—	—	1	91
	Female ...	4	8	11	22	18	5	10	5	6	6	4	2	2	2	1	1	—	1	1	1	16	126
	Total ...	6	13	26	33	28	15	22	11	13	7	6	5	6	3	1	1	—	2	1	1	17	217
Recoveries	Male ...	—	1	1	2	—	2	1	—	3	2	—	—	1	—	1	—	—	—	—	—	—	14
	Female ...	1	2	2	3	4	1	1	—	2	—	2	2	1	1	—	—	—	—	—	—	12	34
	Total ...	1	3	3	5	4	3	2	—	5	2	2	2	2	1	1	—	—	—	—	—	12	48
Grand Total		7	16	29	38	32	18	24	11	18	9	8	7	8	4	2	1	—	2	1	1	29	265

Table II.—Meningitis cases*

Amongst those reported as Vomiting Sickness.

		Under 1 year	1-2 years	2-3 years	3-4 years	4-5 years	5-6 years	6-7 years	7-8 years	8-9 years	9-10 years	10-11 years	11-12 years	12-13 years	13-14 years	14-15 years	15-16 years	16-17 years	17-18 years	18-19 years	19-20 years	Over 20 years	Total
Fatal Cases	Male ...	—	1	2	3	2	3	1	4	2	—	1	—	2	1	—	—	—	1	—	—	—	23
	Female ...	4	1	2	4	3	1	2	—	1	2	—	—	—	—	—	1	—	—	—	1	1	23
	Total ...	4	2	4	7	5	4	3	4	3	2	1	—	2	1	—	1	—	1	—	1	1	46
Recoveries	Male ...	—	—	1	—	—	—	1	—	1	—	—	—	—	—	1	—	—	—	—	—	—	4
	Female ...	1	—	1	—	—	—	—	—	1	—	—	2	—	—	—	—	—	—	—	—	1	6
	Total ...	1	—	2	—	—	—	1	—	2	—	—	2	—	—	1	—	—	—	—	—	1	10
Grand Total		5	2	6	7	5	4	4	4	5	2	1	2	2	1	1	1	—	1	—	1	2	56

* That is, cases exhibiting symptoms of Meningitis, and in whose spinal fluid a Gram-negative diplococcus was found.

TABLE III.—Vomiting Sickness less 'Meningitic'* cases.

		Under 1 year	1-2 years	2-3 years	3-4 years	4-5 years	5-6 years	6-7 years	7-8 years	8-9 years	9-10 years	10-11 years	11-12 years	12-13 years	13-14 years	14-15 years	15-16 years	16-17 years	17-18 years	18-19 years	19-20 years	Over 20 years	Total
Fatal Cases	Male ...	2	4	13	8	8	7	11	2	5	1	1	3	2	—	—	—	—	—	—	—	1	68
	Female ...	—	7	9	18	15	4	8	5	5	4	4	2	2	2	1	—	—	1	1	—	15	103
	Total ...	2	11	22	26	23	11	19	7	10	5	5	5	4	2	1	—	—	1	1	—	16	171
Recoveries	Male ...	—	1	—	2	—	2	—	—	2	2	—	—	1	—	—	—	—	—	—	—	—	10
	Female ...	—	2	1	3	4	1	1	—	1	—	2	—	1	1	—	—	—	—	—	—	11	28
	Total ...	—	3	1	5	4	3	1	—	3	2	2	—	2	1	—	—	—	—	—	—	11	38
Grand Total		2	14	23	31	27	14	20	7	13	7	7	5	6	3	1	—	—	1	1	—	27	209

* For details as to age, etc., see Table II.

TABLE IV.—Vomiting Sickness,* after deduction of Meningitis and 'probable Meningitis' cases.

		Under 1 year	1-2 years	2-3 years	3-4 years	4-5 years	5-6 years	6-7 years	7-8 years	8-9 years	9-10 years	10-11 years	11-12 years	12-13 years	13-14 years	14-15 years	15-16 years	16-17 years	17-18 years	18-19 years	19-20 years	Over 20 years	Total
Fatal Cases	Male ...	2	4	13	6	7	7	11	2	4	1	1	2	2	—	—	—	—	—	—	—	1	63
	Female ...	—	7	7	15	13	4	8	4	5	4	3	2	2	2	1	—	—	1	1	—	15	94
	Total ...	2	11	20	21	20	11	19	6	9	5	4	4	4	2	1	—	—	1	1	—	16	157
Recoveries	Male ...	—	1	—	2	—	2	—	—	2	1	—	—	1	—	—	—	—	—	—	—	—	9
	Female ...	—	2	1	3	4	1	1	—	1	—	2	—	1	1	—	—	—	—	—	—	11	28
	Total ...	—	3	1	5	4	3	1	—	3	1	2	—	2	1	—	—	—	—	—	—	11	37
Grand Total		2	14	21	26	24	14	20	6	12	6	6	4	6	3	1	—	—	1	1	—	27	194

* Six of these were probable only (see text).

TABLE V.

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
1	B.A.M. ...	2	F.	–	–	...	...	+	+	...	...	+	–	+	–	–	–	D.	5 hours	
2	C.R. ...	3	M.	–	–	...	...	+	+	...	...	+	–	+	–	–	–	D.	15 ,,	
3	L.H. ...	18	F.	–	–	+	+	...	+	...	...	+	–	+	–	–	–	D.	20 ,,	
4	M.H. ...	8	M.	–	+	+	+	...	–	...	...	+	–	+	–	–	–	D.	36 ,,	
5	M.J. ...	$1\frac{5}{12}$	F.	–	–	...	...	+	?	...	...	+	–	+	–	–	–	D.	2 ,,	Slight twitchings, no general convulsions
6	L.M. ...	4	F.	–	–	–	+	...	+	...	...	+	101° F.	+	–	–	–	D.	3 ,,	
7	—.R. ...	$3\frac{1}{2}$	F.	...	...	...	...	...	+	...	...	+	–	+	–	–	–	D.	7 ,,	'Vomiting Sickness without vomiting'
8	E.C. ...	6	F.	...	–	+	+	...	+	...	...	+	–	+	–	–	–	D.	7 ,,	
9	B.C. ...	$1\frac{3}{12}$	F.	...	...	+	–	...	...	...	...	...	–	+	–	–	–	R.	...	Vomiting only, others in family typical
10	C.N. ...	4	M.	–	–	...	+	...	+	...	...	+	–	+	–	–	–	D.	5 hours	
11	L.W. ...	6	F.	...	...	...	+	...	+	...	...	+	–	+	–	–	–	D.	4 ,,	
12	P.H. ...	$2\frac{10}{12}$	M.	–	–	...	...	+	...	...	...	+	–	+	–	–	–	D.	15 ,,	No mention of convulsions
13	L.McE. ...	25	F.	...	...	+	+	...	?	...	...	+	–	+	–	–	–	D.	...	Duration not stated, comatose when seen
14	L.H. ...	25	F.	–	–	...	...	+	+	...	...	+	–	+	–	–	–	D.	12 hours	
15	M.W. ...	$1\frac{2}{12}$	F.	–	?	+	+	...	+	...	...	+	–	+	–	–	–	D.	10 ,,	Appeared to have abdominal pain, but not certain
16	S.M.S. ...	$3\frac{6}{12}$	F.	–	...	...	...	+	+	...	...	+	Slight	+	–	–	–	D.	12 ,,	'Slight fever,' temperature not stated
17	C.T. ...	8	M.	...	...	...	...	+	+	...	...	+	–	+	–	–	–	D.	5 ,,	
18	A.G. ...	$3\frac{1}{2}$	F.	...	...	...	...	...	+	...	...	+	...	+	–	–	–	D.	8 ,,	No mention of vomiting; p.m. typical
19	A.L. ...	6	F.	...	...	...	...	+	?	...	...	+	–	+	–	–	–	D.	8 ,,	No definite convulsions
20	J.E.B. ...	$3\frac{1}{2}$	M.	...	+	+	+	...	+	–	–	+	–	+	–	–	–	D.	44 ,, 8 ,,	Prolonged quiescent interval, 33 hours
21	D.G. ...	3	M.	–	...	+	+	...	+	...	...	+	–	+	–	–	–	D.	10 ,,	
22	W.S. ...	7	M.	–	– ?	+	...	...	...	...	...	+	...	+	–	–	–	D.	3 ,,	Abdominal discomfort, no real pain
23	F.P. ...	8	M.	...	...	+	...	...	+	...	...	+	–	+	–	–	–	D.	6 ,,	No secondary vomiting
24	M.H. ...	17	F.	...	...	+	+	...	+	–	–	+	–	+	–	–	–	D.	12 ,,	
25	L.M. ...	5	F.	...	...	...	...	+	+	...	...	+	...	+	–	–	–	D.	10 ,,	
26	M.A.P. ...	25	F.	–	+	+	–	...	–	...	...	+	–	+	–	–	–	D.	? 26 ,,	Lapsed into coma after initial vomiting, no convulsions noticed
27	L.T. ...	8	F.	...	...	+	+	...	?	–	–	+	–	+	–	–	–	D.	24 ,,	Twitching only, no general convulsions
28	S.R. ...	$6\frac{1}{2}$	M.	...	...	...	...	...	+	–	?	+	–	+	–	–	–	D.	4 ,,	Convulsions and coma only mentioned; rigidity of neck only during convulsions
29	L.E. ...	6	M.	...	...	...	...	+	...	–	–	+	–	+	–	–	–	D.	2 ,,	Coma soon after vomiting; (no 'initial' vomiting)
30	H.C. ...	37	F.	...	+	+	–	...	–	–	–	–	–	+	–	–	–	R.	...	Child of this one died. Mother vomiting and recovered
31	P.W. ...	6	M.	–	+	...	+	...	+	...	...	+	–	+	–	–	–	D.	2 hours	
32	D.C. ...	11	M.	–	+	+	+	...	+	...	...	+	–	+	–	–	–	D.	20 ,,	

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
33	C.L. ...	1	F.	...	...	...	...	+	+	...	...	?	−	+	−	−	−	D.	¼ hour	History uncertain, became so acute
34	L.R. ...	2	M.	...	...	...	+	...	+	−	−	+	−	+	−	−	−	D.	2 hours	
35	O.F. ...	25	F.	−	−	+	+	...	+	−	−	+	−	+	−	−	−	D.	12 ,,	
36	—.F. ...	4	F.	−	−	+	+	...	+	−	−	+	−	+	−	−	−	D.	4 ,,	
37	M. ...	3	F.	...	...	...	...	+	+	−	−	+	−	+	−	−	−	D.	6½ ,,	
38	L.W. ...	21	F.	...	...	−	−	−	+	−	−	+	−	+	−	−	−	D.	2 ,,	'Vomiting Sickness without vomiting'
39	S.N. ...	2½	M.	−	−	+	?	...	?	...	...	+	−	+	−	−	−	D.	? 38 ,,	History incomplete
40	K.L.W. ...	2	F.	...	...	−	−	...	+	...	...	+	−	+	−	−	−	D.	3 ,,	'Vomiting Sickness without vomiting'
41	J.W. ...	2½	M.	...	...	+	+	...	+	−	−	+	−	+	−	−	−	D.	3 ,,	
42	A.L. ...	7	M.	+	−	+	+	...	+	...	...	+	..	−	−	+	−	D.	21 ,,	Diplococcus present
43	C.D. ...	1	F.	+	−	At intervals throughout			+	?	+	+	107°	−	−	+	−	D.	? 1 week	Diplococcus present
44	H.B. ...	3	M.	...	...	...	...	...	+	...	+	+	+	−	−	+	−	D.	? 2 weeks	Diplococcus present
45	S.C. ...	17	M.	+	−	+	...	...	...	...	?	+	+	−	−	+	−	D.	?	Diplococcus present; ill on and off for a month
46	R.P. ...	3½	F.	+	−	+	+	...	+	+	+	+	...	−	−	+	−	D.	9 hours	Diplococcus present
47	C.P. ...	8	M.	+	−	+	+	...	+	+	+	+	...	−	−	+	−	D.	10 ,,	Diplococcus present
48	E.P. ...	15½	F.	+	−	+	+	...	+	+	+	+	...	−	−	+	−	D.	12 ,,	Diplococcus present
49	V.P. ...	11	F.	+	−	+	...	...	−	+	+	−	?	−	−	+	−	R.	...	Diplococcus present
50	O.S. ...	6	M.	+	−	+	+	...	+	+	+	−	Slight	−	−	+	−	R.	...	Diplococcus present; ill for several days
51	P.McM. ...	4	F.	+	−	...	...	...	+	+	+	+	...	−	−	+	−	D.	8 hours	Diplococcus present
52	A.P. ...	2	F.	+	−	+	...	...	−	+	+	−	99·4°	−	−	+	−	R.	...	Diplococcus present
53	E.P. ...	9/12	F.	+ ?	−	+	...	...	−	?	+	−	...	−	−	+	−	R.	...	Diplococcus present
54	V.P. ...	8	F.	+	−	+	...	...	−	+	+	−	100°	−	−	+	−	R.	...	Diplococcus present
55	H.P. ...	25	F.	+	−	+	...	...	−	−	−	−	...	+	−	−	−	R.	...	Vomiting and headache only, but reported as Vomiting Sickness
56	J.L.B. ...	4	M.	−	+	+	+	...	+	−	−	+	−	+	−	−	−	D.	2 hours	
57	L.H.G. ...	3/12	M.	−	+	?	...	...	+	−	−	+	...	+	−	−	−	D.	½ hour	History indefinite, very acute
58	M.B. ...	2 8/12	F.	−	−	+	+	...	...	−	−	+	...	+	−	−	−	D.	8½ hours	

All of one family, the 'Peart' series, see text

One family; attended hospital afterwards for some time; others died at home

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
59	E.H. ...	10	F.	+	−	−	−	...	...	+?	+	...	+	−	−	+	−	D.	15 days	Diplococcus present. Convulsions not stated
60	C.M. ...	13	M.	...	...	Throughout		...	+	?	...	+	...	−	−	+	−	D.	11 hours	Diplococcus present
61	C.M.A. ...	4	F.	...	...	Throughout		...	?	+?	+?	+	...	−	−	+	−	D.	15 ,,	Diplococcus present
62	H.W. ...	6	F.	−	−	+	...	...	...	...	...	+	...	+	−	−	−	D.	4 ,,	
63	A.J. ...	2	M.	−	−	+	+	...	−	−	−	+	−	+	−	−	−	D.	6 ,,	No convulsions
64	J.H. ...	3	M.	−	+	...	...	...	?	−	−	...	−	+	−	−	−	D.	15 ,,	
65	O.D. ...	6	F.	...	...	+	?	...	...	...	...	+	...	+	−	−	−	D.	? 24 ,,	History incomplete ; comatose when seen
66	S.A.H. ...	3	F.	−	+	+	+	...	+	...	...	+	−	+	−	−	−	D.	? 24 ,,	
67	R.T. ...	6	F.	?	...	At intervals throughout			+	+	+	+	...	−	−	+	−	D.	18½ ,,	Diplococcus present
68	D.D. ...	11	M.	+	−	At intervals throughout			+	...	+	+	...	−	−	...	+	D.	48 ,,	
69	L.P. ...	5	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	9¼ ,,	
70	A.S. ...	2	F.	...	...	−	−	...	+	...	...	+	−	+	−	−	−	D.	3 ,,	No vomiting
71	R.C.C. ...	8	F.	−	−	At intervals throughout			+	...	+	+	...	−	−	+	−	D.	15 ,,	Diplococcus present
72	G.McL. ...	4	F.	−	−	+?	−	...	?	−	−	−	−	+	−	−	−	R.	...	
73	J.P. ...	4	F.	...	...	−	−	...	+	−	−	+	...	+	−	−	−	D.	6 hours	
74	J.A.D. ...	9	F.	−	+	+	...	...	+	−	−	+	−	+	−	−	−	D.	13 ,,	No secondary vomiting recorded
75	J.H. ...	1 10/12	F.	...	...	+	...	...	...	...	...	...	...	+	−	−	−	D.	½ ,,	Very acute, vomiting only recorded
76	A.C. ...	5½	F.	...	...	...	...	+	+	...	...	+	...	+	−	−	−	D.	12 ,,	
77	M.A. ...	4	F.	...	...	+	+	...	+	...	...	+	100°	+	−	−	−	D.	12 ,,	
78	A.P.D. ...	8	F.	...	...	...	...	+	+	...	...	+	...	+	−	−	−	D.	10 ,,	
79	G.M.F. ...	5	F.	...	...	Throughout		...	+	...	+	+	...	−	−	+	−	D.	8 ,,	Diplococcus present
80	O.B. ...	3	M.	...	...	Throughout		...	−	?	?	+	...	−	−	+	−	D.	? 4 ,,	Meningitis p.m.
81	Z.E.J. ...	7	M.	...	...	Throughout		...	+	...	+	+	?	−	−	+	−	D.	12 ,,	Diplococcus present
82	C.R. ...	5	F.	...	...	+	+	...	+	...	+	+	...	−	−	+	−	D.	3 days	Diplococcus present
83	M.R. ...	7	F.	...	...	+	+	...	+	...	+	+	...	−	−	+	−	D.	14 hours	Diplococcus present
84	I.V. ...	5	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	7 ,,	
85	B.D. ...	4	F.	...	...	−	−	...	+	−	−	+	...	−	+	−	−	D.	3 ,,	Diplococcus present, see text
86	V.C. ...	2	F.	...	...	Throughout		...	...	...	...	...	...	−	−	+	−	D.	½ ,,	Diplococcus present. Vomiting too acute to notice other symptoms
87	E.S. ...	6	F.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	11 ,,	
88	L.F. ...	4	F.	...	...	+	+	...	+	...	...	...	...	+	−	−	−	D.	3 ,,	
89	A.J. ...	6	M.	...	...	+	+	...	?	−	−	...	...	+	−	−	−	D.	23 ,,	
90	J.J. ...	4	M.	...	...	+	+	...	?	−	−	...	...	+	−	−	−	D.	25 ,,	One family; two younger died, two older recovered
91	M.J. ...	10	F.	...	...	+	−	...	−	−	−	−	Slight	+	−	−	−	R.	...	
92	G.J. ...	8	M.	...	...	+	−	...	−	−	−	−	Slight	+	−	−	−	R.	...	

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
93	J.H. ...	3	F.	...	...	...	...	+	+	...	...	+	...	+	−	−	−	D.	10 hours	
94	A.H. ...	4	M.	...	...	Throughout		...	+	+	+	+	...	−	−	+	−	D.	9½ ,,	Diplococcus present
95	S.B. ...	3	F.	...	...	Throughout		...	+	...	+	+	...	−	−	+	−	D.	1½ ,,	Diplococcus present
96	J.F. ...	5	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	12 ,,	
97	R.F. ...	9	F.	...	...	At intervals throughout			...	...	+	+	...	−	−	+	−	D.	36 ,,	Diplococcus present
98	V.L.C. ...	4	F.	...	...	−	−	...	+	...	+	+	+	−	−	...	+	D.	24 ,,	'Typical C.S. Meningitis p.m.,' see text
99	V.F. ...	2	M.	...	...	+	+	...	+	...	...	...	...	+	−	−	−	D.	17 ,,	
100	J.W. ...	12	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	8 ,,	
101	D.W. ...	10	F.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	25 ,,	
102	A.R. ...	24	F.	...	...	+	+	...	?	...	...	?	...	+	−	−	−	D.	8 ,,	
103	C.R. ...	3	F.	...	...	+	+	...	?	...	...	?	...	+	−	−	−	D.	18 ,,	
104	L.J.R. ...	3 8/12	F.	?	−	+	+	...	+	...	...	+	...	+	−	−	−	D.	14 ,,	
105	J.G. ...	8	M.	...	...	Throughout		...	+	...	...	+	...	−	−	...	+	D.	10 ,,	Diplococcus present
106	H.S. ...	3½	F.	...	...	...	...	...	...	...	...	...	...	−	−	...	+	D.	...	Diplococcus present; no history sent
107	A.S. ...	3½	M.	...	...	+	−	...	−	−	−	−	−	+	−	−	−	R.	...	
108	S.S. ...	3	F.	...	...	+	−	...	−	−	−	−	−	+	−	−	−	R.	...	
109	A.W. ...	1 3/12	F.	...	...	+	−	...	−	−	−	−	−	+	−	−	−	R.	...	
110	F.B. ...	1 6/12	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	14 hours	
111	E.M. ...	3½	F.	...	...	Throughout		...	+	...	...	+	...	−	−	...	+	D.	12 ,,	Diplococcus present, see text
112	J.B. ...	5	F.	+	+ ?	+	+	...	+	...	?	−	+	−	−	+	−	D.	?	Diplococcus present
113	E.S. ...	7	M.	...	...	...	...	+	...	...	...	...	...	+	−	−	−	D.	23 hours	Vomiting only symptom mentioned
114	M.C. ...	3 4/12	F.	?	−	See remarks		...	+	...	+	+	...	−	−	...	+	D.	? 4 ,,	History of M., but no specimens sent
115	L.F. ...	4 10/12	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	6 ,,	
116	B.W. ...	12	M.	...	...	+	+	...	+	−	?	+	+	−	−	+	−	D.	3 days	Diplococcus present
117	M.H. ...	27	F.	+ ?	+	+	+	...	−	−	−	+	−	+	−	−	−	D.	2½ ,,	
118	J.J. ...	5	M.	...	...	+	+	...	...	...	...	...	...	+	−	−	−	D.	2 ,,	Recurrent vomiting only mentioned
119	D.C. ...	9/12	F.	...	...	Throughout		...	+	+	+	+	?	−	−	+	−	D.	24 hours	Diplococcus present
120	E.W. ...	35	F.	+	...	+	+	...	−	−	−	+	−	+	−	−	−	D.	?	No convulsions seen
121	E.W. ...	60	F.	−	...	+	+	...	...	...	...	+	−	+	−	−	−	D.	?	No convulsions seen
122	T.C. ...	2¼	F.	...	...	+	+	...	+	...	?	+	−	+	−	−	−	D.	14 hours	
123	J.H. ...	6	F.	+	?	+	+	...	−	...	...	+	...	+	−	−	−	D.	12 ,,	No convulsions seen
124	F.P. ...	3	F.	...	...	+	+	...	+	...	+	+	...	...	+	−	−	D.	12 ,,	Diplococcus present, see text
125	J.B. ...	3	F.	...	...	+	+	...	...	...	...	...	...	+	−	−	−	D.	4 ,,	Vomiting only mentioned in history
126	K.C. ...	3	F.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	9 ,,	
127	J.E.S. ...	1 10/12	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	17 ,,	
128	A.B. ...	3	F.	...	...	+ ?	+	...	+	...	...	+	...	+	−	−	−	D.	1 ,,	
129	Z.P. ...	4	F.	...	...	At intervals throughout			+	...	+	+	...	−	−	+	−	D.	24 ,,	Diplococcus present

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
130	G.A.M. ...	9	F.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	11 hours	
131	D.T. ...	3½	F.	—	+	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
132	W.T. ...	5	M.	—	+	+	—	...	—	+?	—	—	—	+	—	—	—	R.	...	
133	G.A. ...	4½	F.	—	+	+	—	...	—	—	?	—	99°	+	—	—	—	R.	...	
134	J.A. ...	9	M.	+		+	...	...	—	+	+	—	100°	—	—	...	+	R.	...	
135	A.S. ...	1½	M.	+	?	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
136	J.S. ...	3	F.	—	+	+	—	...	—	+	+	—	?	+	—	+	—	R.	...	
137	A.T. ...	14	M.	+	—	At intervals		...	—	+	+	—	?	—	—	+	—	R.	...	Diplococcus present } Brothers
138	V.T. ...	8	M.	+	—	At intervals		...	—	?	+	—	—	—	—	+	—	R.	...	Diplococcus present }
139	R.H. ...	8	M.	—	—	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
140	L.M. ...	1	F.	...	...	+	...	...	—	...	?	—	—	+	—	—	—	R.	...	
141	R.E. ...	-	M.	+	-	Throughout		...	+	+	+	+	...	—	—	+	—	D.	?	Diplococcus present
142	M.F. ...	Adult	F.	+	—	Throughout		...	+	+	+	+	+	—	—	+	—	D.	?	Diplococcus present
143	R.T. ...	11	F.	+	—	At intervals		...	...	?	?	—	101°	—	—	+	—	R.	...	Diplococcus present
144	M.S. ...	Adult	F.	—	—	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
145	J.McL. ...	2½	F.	—	—	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
146	L.M. ...	12	F.	...	...	+	—	...	—	...	...	—	—	+	—	—	—	R.	...	
147	W.P. ...	5½	M.	+	?	At intervals		...	—	+	?	...	102°	—	—	+	—	R.	...	
148	A.M. ...	5	F.	?	—	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
149	D.W. ...	3½	M.	+	-	Throughout		...	+	+	?	+	...	—	—	+	—	D.	? 12 hours	Diplococcus present
150	J.W. ...	4	F.	...	...	—?	—	...	+	...	...	+	...	+	—	—	—	D.	?	
151	G.F. ...	5	F.	—	—	+	+	...	+	...	...	+	...	+	—	—	—	D.	8 hours	
152	C.B. ...	3	M.	...	...	+	+	...	...	...	...	+	...	+	—	—	—	D.	5 ,,	No mention of convulsions
153	C.N. ...	5½	M.	...	...	+	+	...	—	—	—	+	...	+	—	—	—	D.	?	
154	A.N. ...	12	M.	—	—	+	—	...	—	...	...	—	...	+	—	—	—	R.	...	
155	C.N. ...	9	M.	—	—	+	+	...	—	...	...	+	...	+	—	—	—	D.	20 hours	
156	E.B. ...	6	M.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	18 ,,	
157	L.A.M. ...	3	M.	...	...	+	—	...	—	...	...	—	...	+	—	—	—	R.	...	
158	G.B. ...	1 1/12	F.	—	?	—	+	...	+	...	...	+	...	+	—	—	—	D.	37 hours	
159	E.B. ...	2	M.	...	...	...	...	...	...	+?	+	+	...	—	—	+	—	D.	?	Diplococcus present
160	H.M. ...	1 7/12	M.	...	...	At intervals		...	+	...	+	+	...	—	—	+	—	D.	50 hours	Diplococcus present
161	A.F. ...	25	F.	+	—	+	—	—	?	—	—	—	—	+	—	—	—	R.	...	
162	C.C. ...	2	M.	...	...	...	...	+	...	...	...	+	—	+	—	—	—	D.	?	No mention of convulsions
163	P.B. ...	11	M.	...	...	+	+	...	—	...	...	...	...	+	—	—	—	D.	33 hours	
164	E.M. ...	11	F.	...	...	+	+	...	+	...	...	+	Slight	+	—	—	—	D.	25 ,,	
165	H.P. ...	2	M.	...	...	—	—	...	+	...	...	+	...	+	—	—	—	D.	6 ,,	? 'Vomiting Sickness, without vomiting'
166	D.D. ...	12	M.	...	...	At intervals throughout			+	?	+	+	...	—	—	+	—	D.	14½ ,,	Diplococcus present

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
167	D.P. ...	4	M.	—	+	At intervals throughout			+	...	+	+	...	—	—	+	—	D.	14 hours	Diplococcus present
168	N.M. ...	8	M.	...	...	+	+	...	...	...	...	+	...	—	—	+	—	D.	33 ,,	Diplococcus present
169	C.G. ...	5	M.	...	...	+	...	...	+	...	...	+	...	+	—	—	—	D.	8 ,,	No secondary vomiting
170	C.B. ...	4	M.	...	...	At intervals throughout			+	...	+	+	...	—	—	...	+	D.	14 ,,	Probably M., no specimens sent
171	I.S. ...	2	F.	...	...	—	—	...	+	+	+	+	+	—	—	...	+	D.	12 ,,	Probably M., no specimens sent
172	I.B. ...	40	F.	...	...	+	+	...	?	...	...	...	...	+	—	—	—	D.	27 ,,	
173	E.M.Y. ...	$2\frac{1}{2}$	F.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	5 ,,	
174	P.B. ...	4	F.	...	...	At intervals throughout			+	...	+	+	...	—	—	...	+	D.	16 ,,	Probably M., no specimens sent
175	E.McD. ...	6	M.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	$3\frac{1}{2}$,,	
176	G.McD. ...	8	F.	...	...	+	+	...	—	...	...	+	...	+	—	—	—	D.	40 ,,	Lapsed into coma without convulsions
177	L.L. ...	12	F.	...	...	+	+	...	?	...	...	+	...	+	—	—	—	D.	3 days	
178	P.H. ...	13	F.	...	...	+	—	...	—	...	...	—	...	+	—	—	—	R.	...	
179	M.B. ...	3	M.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	?	
180	C.W. ...	$2\frac{1}{2}$	F.	...	...	Throughout		...	+	+	...	+	...	—	—	...	+	D.	$4\frac{1}{2}$ hours	Diplococcus present, atypical
181	C.B. ...	$2\frac{7}{12}$	F.	...	...	At intervals throughout			+	?	...	+	...	—	—	+	—	D.	?	Diplococcus present
182	G.T. ...	5	M.	...	...	?	+	...	+	?	...	...	...	+	—	—	—	D.	13 hours	Reported as Vomiting Sickness, but history uncertain
183	E.P. ...	2	M.	...	...	Throughout		...	+	...	...	+	...	—	—	+	—	D.	? 3 ,,	Diplococcus present
184	E.A.W. ...	$1\frac{10}{12}$	F.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	$3\frac{1}{2}$,,	
185	T.E. ...	13	F.	...	...	+	+	...	—	...	...	+	...	+	—	—	—	D.	$7\frac{1}{2}$,,	Vomiting to coma, no convulsions
186	M.B. ...	4	F.	...	...	+	+	...	+	...	+	+	...	—	+	—	—	D.	11 ,,	Diplococcus seen, but no growth. Symptoms of Vomiting Sickness
187	B.M. ...	$\frac{11}{12}$	F.	...	...	Throughout		...	+	...	+	+	...	—	—	+	—	D.	? 2 ,,	Diplococcus present
188	E.A. ...	3	F.	...	...	—	—	...	+	...	+	+	...	—	?	—	—	D.	8 ,,	Reported as Vomiting Sickness, ? Why, see text
189	T.J. ...	12	M.	...	...	...	...	...	...	...	...	...	...	—	—	+	—	D.	?	Diplococcus present, but no history sent
190	T.T. ...	$2\frac{5}{12}$	M.	...	...	—	—	...	+	...	...	+	...	+	—	—	—	D.	$1\frac{3}{4}$ hours	'Vomiting Sickness, without vomiting'
191	J.S. ...	30	F.	...	+	+	—	...	—	...	...	—	99°	+	—	—	—	R.	...	
192	B.T. ...	4	F.	...	...	+	—	...	—	...	...	—	—	+	—	—	—	R.	...	
193	J.T. ...	5	M.	...	...	+	+	...	?	...	...	+	...	+	—	—	—	D.	24 hours	'No definite convulsions'
194	J.E. ...	6	F.	...	...	—	—	...	+	...	+	+	...	—	?	—	—	D.	$22\frac{1}{2}$,,	No specimens sent. See text
195	M.A.W. ...	$3\frac{3}{12}$	F.	...	...	+	+	...	+	—	—	+	—	+	—	—	—	D.	$14\frac{3}{4}$,,	
196	M.R. ...	7	F.	...	...	...	...	...	+	...	...	+	...	+	—	—	—	D.	2 ,,	History incomplete
197	J.E. ...	$6\frac{1}{2}$	M.	...	...	—	—	...	+	...	...	+	...	+	—	—	—	D.	24 ,,	No vomiting
198	E.E. ...	20	F.	...	...	?	...	...	+	...	...	+	+	+	—	—	—	D.	24 ,,	

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
199	D.A. ...	7	F.	...	+	...	...	...	+	...	...	+	−	+	−	−	−	D.	15 hours	No history of vomiting
200	A.B. ...	5	M.	...	...	−	−	...	+	...	+	+	...	−	−	+	−	D.	6 ,,	Diplococcus present
201	E.P. ...	27	F.	...	...	+	+	...	...	...	...	...	...	+	−	−	−	D.	9 ,,	History incomplete
202	F.E. ...	3	M.	...	...	Throughout		...	+	...	+	+	...	−	−	+	−	D.	? 2 ,,	Diplococcus present
203	A.T. ...	6	M.	...	...	At intervals throughout			+	...	+	+	...	−	−	+	−	D.	48 ,,	Diplococcus present
204	B.	$2\frac{1}{2}$	...	...	...	...	...	...	...	...	...	...	...	+	−	−	−	D.	?	Reported as Vomiting Sickness.
205	T.E.S. ...	4	F.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	8 hours	Details lost
206	C.W. ...	30	F.	...	+	+	+	...	+	...	...	+	...	+	−	−	−	D.	12 ,,	
207	M.C. ...	$\frac{11}{12}$	F.	...	...	At intervals throughout			+	...	+	+	102°	−	−	+	−	D.	2 days	Diplococcus present
208	W.D. ...	$2\frac{11}{12}$	M.	...	...	Throughout		...	+	...	...	...	...	−	−	...	+	D.	?	Diplococcus present, history
209	C.W. ...	12	M.	...	+	...	...	...	?	...	...	+	...	+	−	−	−	D.	$2\frac{1}{2}$ days	incomplete
210	M.G. ...	7	F.	...	...	+	+	...	+	...	...	...	...	+	−	−	−	D.	6 hours	
211	E.G. ...	10	F.	...	...	Throughout		...	+	...	...	...	...	−	−	...	+	D.	9 ,,	Diplococcus present, history incomplete
212	E.H. ...	9	F.	+	−	At intervals throughout			?	...	...	+	...	−	−	+	−	D.	30 ,,	Diplococcus present
213	E.M. ...	$\frac{11}{12}$	F.	...	...	Throughout		...	+	...	...	+	...	−	−	+	−	D.	3 ,,	Diplococcus present
214	R.A.L. ...	9	F.	...	...	...	...	...	+	...	...	+	...	+	−	−	−	D.	6 ,,	Vomiting not stated, reported as Vomiting Sickness
215	C.T. ...	$2\frac{1}{2}$	M.	...	...	−	−	...	+	+	+	−	+	−	−	+	−	R.	...	Symptoms of M., but no specimens sent
216	N.W. ...	7	F.	...	...	?	?	...	+	...	+	+	...	−	−	...	+	D.	?	Diplococcus present
217	D.H. ...	3	M.	...	...	Throughout		...	+	...	...	...	...	−	−	...	+	D.	? 2 hours	Diplococcus present
218	C.G. ...	6	M.	...	...	+	+	...	+	...	...	+	...	?	?	?	?	D.	16 ,,	Diplococcus present. See text
219	C.W. ...	$1\frac{7}{12}$	M.	...	+	+	+	...	+	−	−	+	−	+	−	−	−	D.	1 hour	
220	P.H. ...	$2\frac{1}{2}$	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	16 hours	
221	L.S. ...	4	F.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	11 ,,	
222	C.C. ...	6	M.	...	...	+	+	...	?	...	...	...	...	+	−	−	−	D.	12 ,,	History incomplete
223	L.C. ...	3	F.	...	...	Throughout		...	+	...	...	+	...	−	−	+	−	D.	$6\frac{1}{2}$,,	Diplococcus present. Preceding Catarrh
224	K.R. ...	$\frac{4}{12}$	M.	...	...	+	+	...	−	...	...	+	...	+	−	−	−	D.	?	No convulsions seen
225	L.R. ...	40	F.	+	−	At intervals throughout			?	...	...	+	...	−	−	+	−	D.	1 week	Diplococcus present
226	H.W. ...	4	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	12 hours	
227	J.D. ...	2	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	2 ,,	
228	J.T. ...	4	M.	...	...	+	+	...	+	...	...	+	...	+	−	−	−	D.	4 ,,	
229	G.J. ...	4	M.	+	−	Throughout		...	...	...	...	...	...	+	−	−	−	D.	1 hour	Very acute; other symptoms not
230	I.S. ...	3	F.	...	...	+	+	...	+	...	?	+	...	+	−	−	−	D.	6 hours	noted

TABLE V.—*continued.*

No.	Initials	Age	Sex	Pain		Vomiting			Convulsions	Kernig's Sign	Rigidity of Neck-Muscle	Coma	Fever	Diagnosis				Result	Duration	Remarks
				Head	Abdomen	Initial	Secondary	Time not stated						Vomiting Sickness	? V.S.	Meningitis	? M			
231	I.R. ...	4½	F.	...	+	...	...	...	+	...	...	+	...	+	—	—	—	D.	14 hours	? Vomiting; 6 hours interval
232	M.H. ...	10	F.	...	...	+	+	...	+	...	...	...	?	+	—	—	—	D.	29 ,,	
233	E.D. ...	4	F.	...	+	...	...	...	+	...	...	+	...	+	—	—	—	D.	7 ,,	Vomiting not mentioned
234	S.M. ...	12	F.	...	+	+	+	...	—	...	...	+	...	+	—	—	—	D.	8 ,,	No convulsions seen
235	D.G. ...	6	F.	—	—	+	+	...	+	...	...	+	...	+	—	—	—	D.	33 ,,	Final attack 8 hours; interval of 24 hours
236	N.R.D. ...	10	M.	—	+	+	+	...	+	...	...	+	...	+	—	—	—	D.	19 ,,	
237	M.C. ...	13	F.	—	—	+	+	...	+	...	...	+	...	+	—	—	—	D.	34 ,,	
238	R.C. ...	26	F.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
239	J.J. ...	65	F.	—	+	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
240	B.J. ...	21	F.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
241	G.G. ...	8	M.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	11 hours	
242	D.G. ...	11	F.	...	...	+	+	...	+	...	...	+	...	+	—	—	—	D.	7 ,,	
243	W.G. ...	9	M.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
244	A.G. ...	4	F.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
245	F.W. ...	9	F.	...	...	+	+	...	+	—	—	+	?	+	—	—	—	D.	? 18 hours	
246	H.W. ...	1 9/12	M.	...	+	+	+	...	+	—	—	+	—	+	—	—	—	D.	2 ,,	
247	P.C. ...	25	F.	...	+	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
248	W.B.H. ...	5	M.	—	—	+	+	...	+	—	—	+	—	+	—	—	—	D.	4 hours	
249	G.S.H. ...	2 10/12	M.	...	...	+	+	...	+	—	—	+	—	+	—	—	—	D.	5 ,,	
250	A.M.H. ...	8	F.	...	...	+	+	...	+	—	—	+	—	+	—	—	—	D.	8 ,,	
251	A.H. ...	42	F.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
252	J.McB. ...	6 9/12	M.	—	—	+	+	...	+	—	—	+	—	+	—	—	—	D.	10½ hours	
253	L.R. ...	26	F.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
254	B.R. ...	14 11/12	F.	...	...	+	+	...	?	—	—	+	—	+	—	—	—	D.	48 hours	
255	R.M. ...	4	F.	...	...	—	—	+	+	—	+	+	+	+	—	—	—	D.	4 ,,	No vomiting
256	C.S. ...	25	F.	...	...	+	+	...	+	—	—	+	—	+	—	—	—	D.	25 ,,	
257	E.B. ...	1 1/12	F.	...	...	...	+	...	+	—	—	+	—	+	—	—	—	D.	8 ,,	No history of 'initial' vomiting
258	C.W. ...	8	F.	...	...	+	+	...	—	—	—	+	101·4°	+	—	—	—	D.	44 ,,	No convulsions noticed
259	S.D. ...	6	F.	...	...	+	—	...	—	—	—	—	101°	+	—	—	—	R.	...	
260	J.F. ...	4	F.	...	...	—	—	...	+	—	—	+	—	+	—	—	—	D.	21 hours	'Vomiting Sickness without vomiting'
261	C.Y. ...	10	F.	—	+	+	+	...	+	—	—	+	—	+	—	—	—	D.	19 ,,	
262	E.M. ...	25	F.	...	...	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
263	P.M. ...	6	F.	—	+	+	—	...	—	—	—	—	—	+	—	—	—	R.	...	
264	P.R. ...	8	M.	—	—	+	—	...	+	—	—	+	—	+	—	—	—	D.	20 hours	No 'secondary' vomiting
265	A.R. ...	2 10/12	F.	—	+	+	+	—	+	—	—	+	—	+	—	—	—	D.	4 ,,	

EXPLANATION OF PLATES

Plate I

Drawings reduced to $\frac{3}{4}$ original size for purposes of reproduction.

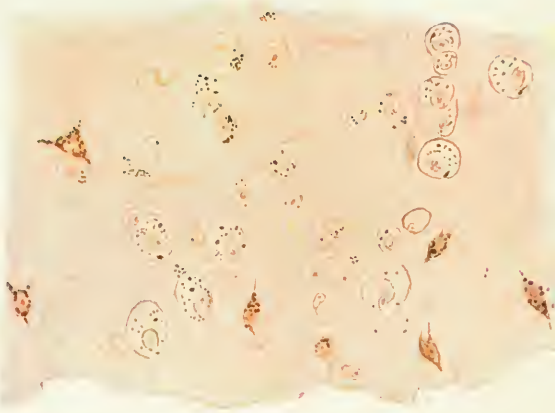
Fig. 1. Cerebrum of dog, after ingestion of ackee extract, Showing minute fat droplets in nerve cells and capillary. Flemming's fixative and safranin stain. 4 mm. objective, comp. oc. 4, tube 150.

Fig. 2. Kitten 3. Liver, after ingestion of ackee extract. Showing fatty metamorphosis, less advanced stage than fig. 3. Flemming and safranin. 4 mm. obj., oc. 4, tube 150.

Fig. 3. Kitten 2. Liver, after ingestion of ackee extract. More advanced fatty degeneration than in Kitten 3 (see last fig.). Flemming and safranin. 4 mm. obj., oc. 4, tube 160.

Fig. 4. Kitten 2. Kidney, after ingestion of ackee extract. Fatty degeneration of ascending limbs of Henle's loop, and of convoluted tubule. Flemming and safranin. 4 mm. obj., oc. 4, tube 160.

Fig. 5. Kitten 2. Kidney, after ingestion of ackee extract. Many tubules have lost epithelium and contain blood, apparent increase of interstitial tissue in consequence of disappearance of epithelium. Stained haematoxylin and Hansen's modification of Van Gieson. 4 mm. obj., oc. 4, tube 158.



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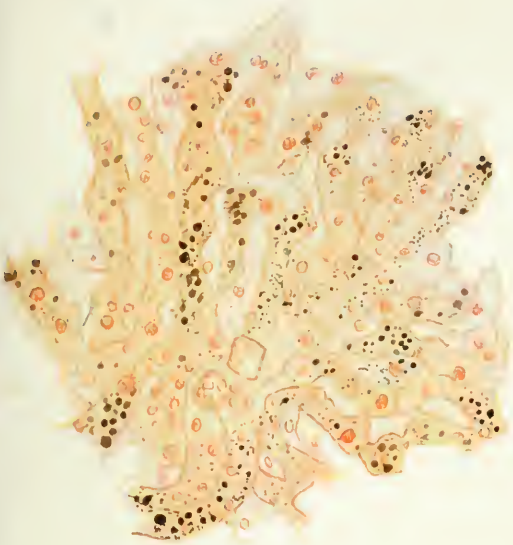

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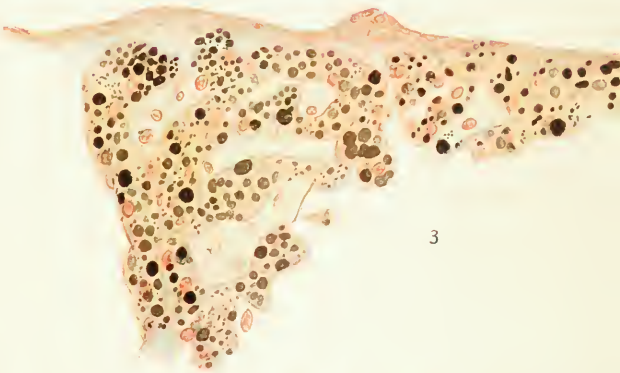

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PATHOLOGY OF VOMITING SICKNESS.

Plate II

Micro-photographs reduced to $\frac{3}{4}$ original size for purposes of reproduction.

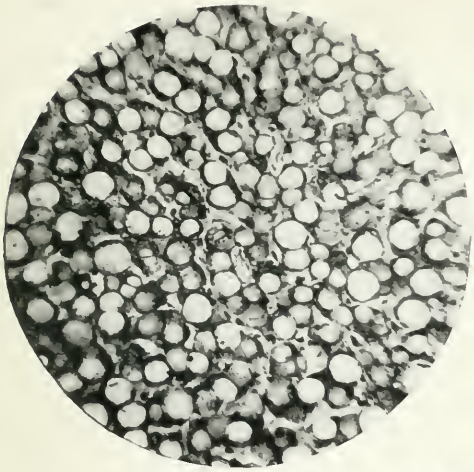
Fig. 1. Liver from the case of ackee poisoning, M.S., described in the text, showing intense fatty metamorphosis. This should be compared with Seidelin's report, Pl. XXXII, fig. 3. The change is even more marked in the case of M.S., because it is the result of repeated small doses of the poison, with a fulminating termination.

Fig. 2. Pancreas from M.S. (ackee poisoning) showing a few acini with recognisable epithelium, but marked necrobiosis and fatty changes elsewhere.

Fig. 3. Liver from E.W., a case of human vomiting sickness, showing fatty changes and necrobiosis. The condition here is intermediate between that of the kitten, fig. 4, and the intense affection of the ackee poisoning case, M.S., fig. 1.

Fig. 4. Kidney from same patient, E.W., showing marked epithelial necrobiosis. It is even more marked than in Seidelin's case (Pl. XXXIII, fig. 4, of his report).

Fig. 5. Pancreas from L.D., another case of vomiting sickness, showing necrobiosis and fatty change almost universally distributed.



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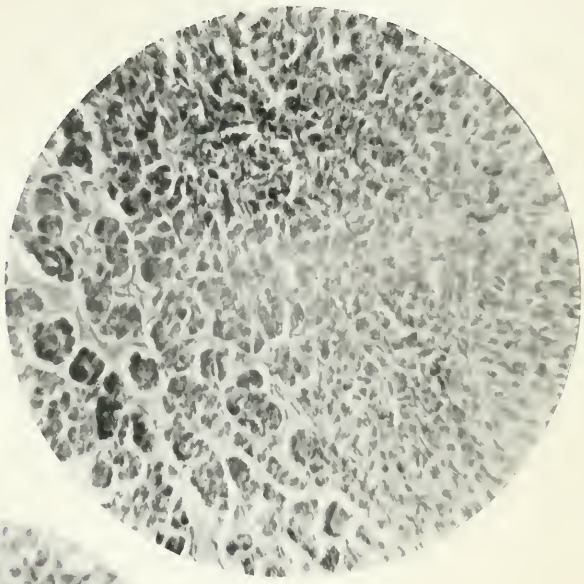

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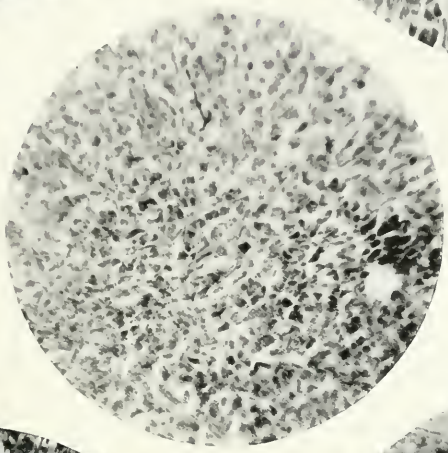

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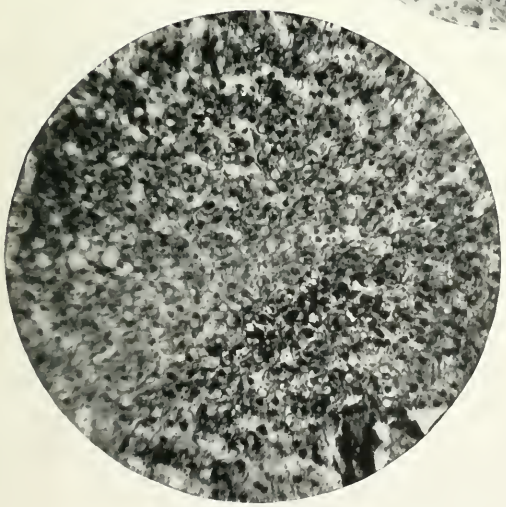

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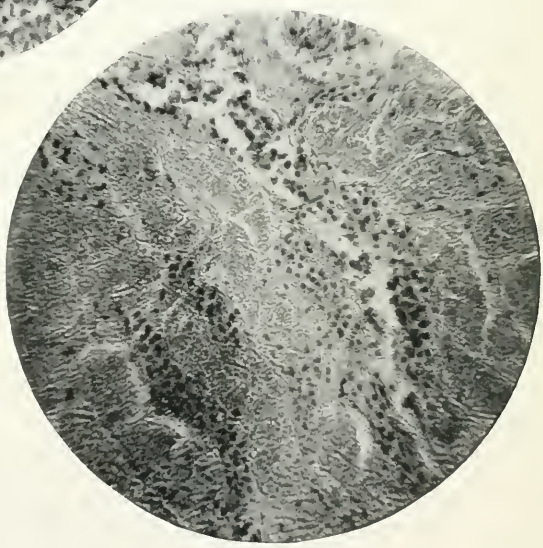

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PATHOLOGY OF VOMITING SICKNESS.